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Chapter 7: Diabetes and the Diabetic Foot

Pamela Scarborough; James McGuire

INTRODUCTION

CHAPTER OBJECTIVES

At the end of this chapter, the learner will be able to:

1. **Discuss the trends in diabetes.**
2. **List the effects of diabetes on all wound etiologies.**
3. **Identify risk factors leading to diabetic foot ulcers.**
4. **Compare and contrast different interventions appropriate for treatment of diabetic foot ulcers.**
5. **Develop a comprehensive plan of care for a patient with a diabetic foot ulcer.**
6. **Select the proper footwear to both prevent and treat the diabetic foot.**

The classic model of the neuropathic foot is most frequently associated with diabetes. Diabetes is a commonly encountered comorbidity in the population of patients with wounds. While many comorbidities have the potential to impact wound healing, this is especially true with diabetes. Therefore, the health care professional needs to understand the diabetes disease process, its implications on the general health of the patient, and its impact on wound healing specifically. The wound care clinician does not manage the diabetes—this responsibility belongs to another provider, usually a primary care physician or an endocrinologist who coordinates the overall diabetes plan of care.

However, it is the responsibility of the wound care clinician to (1) review the patient's blood glucose values to ensure there is adequate control of the disease to support effective wound healing and (2) recognize when the disease may be negatively affecting a patient's wound healing potential.

CLINICAL CONSIDERATION

The disease state of diabetes is managed by a team of specialists. In the absence of a team, the primary care physician or endocrinologist will direct the medical management of this complicated disease.

This chapter provides an overview of the epidemiology of diabetes, its effects on wound healing, clinical challenges that the comorbidity of diabetes poses to the form and function of the foot, and considerations for clinical interventions when a diabetic foot ulcer (DFU) is present. For comprehensive information on the pathophysiology and treatment of diabetes, the reader should consult other sources, including the standards of care from the American Diabetes Association[1] and American Association of Clinical Endocrinologists' comprehensive diabetes management algorithm—2018.[2] This chapter also provides an overview of treatment strategies necessary for patients to control their diabetes in order to assist in optimal treatment of chronic wounds, including diabetic foot ulcers.

DIABETES

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Epidemiology and Health Care Implications

The prevalence of diabetes has been rising steadily and dramatically over the past several decades and is currently considered to be at epidemic proportions worldwide.[1,3] In 2017 the global prevalence for diabetes is estimated to be 425 million individuals, projecting growth to 629 million in 2045.[3] The Centers for Disease Control and Prevention (CDC) estimates that as of 2017 the United States had 30 million people diagnosed with diabetes, or roughly 9.4% of the population, plus another 7 million that are undiagnosed with type 2 diabetes (T2DM).[4] In addition to this number, over 80 million people have the condition of prediabetes, adding up to over 100 million people living with diabetes or prediabetes in the United States. The number of people at risk for or with diabetes continues to increase despite significant efforts to screen and educate the general population about the controllable risks associated with acquiring diabetes: obesity, lack of exercise and physical activity, lack of access to good nutrition, and/or poor dietary choices. These are the major risk factors for T2DM for 90% of the individuals with diabetes, in addition to the strong genetic predisposition to T2DM.

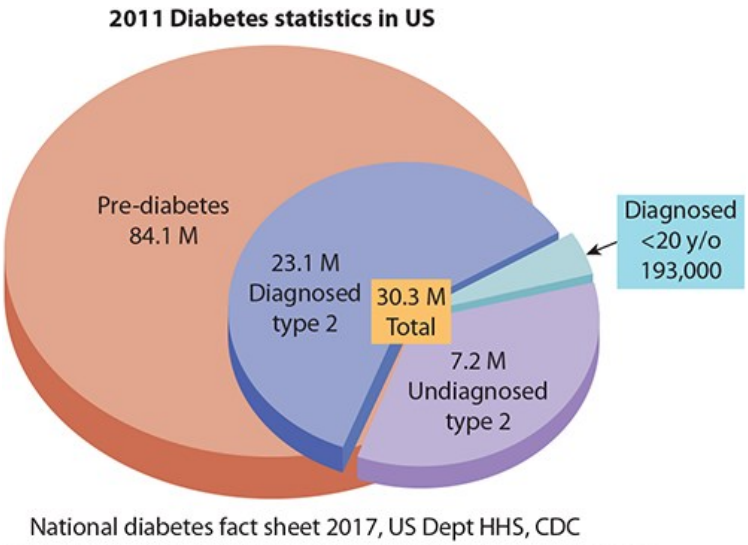
CLINICAL CONSIDERATION

Diabetes is the most costly chronic illness in the United States. One in four health care dollars is spent for someone with the diabetes diagnosis.[5]

The burden of diabetes has profound implications for public health and health care systems both nationally and internationally. The number of people with diabetes in the United States is growing so rapidly that the U.S. health care system is severely challenged financially by this one disease (**FIGURE 7-1**).[4] Currently one in four health care dollars is spent on the management of diabetes and its associated complications.[5,6]

FIGURE 7-1

Diabetes statistics in the United States Statistics on the prevalence of diabetes in the United States show evidence that it is an epidemic disease that accounts for over 30% of the Medicare budget for health care.[4]



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Diabetes has a profound impact on health as a result of secondary complications such as heart disease, kidney disease, retinopathy, neuropathy, chronic wounds, and lower-limb amputation. In addition, people are acquiring T2DM at younger ages than ever before in the United States.[4] Therefore, T2DM is no longer a disease for only the over 40 age group. Accordingly, providers are seeing younger people with diabetes-related complications, including issues related to foot pathology and wound healing challenges. Furthermore, the older population is being affected the most from a number perspective, with the largest prevalence of diabetes in the over 65-year-old age group (**TABLE 7-1**).[4]

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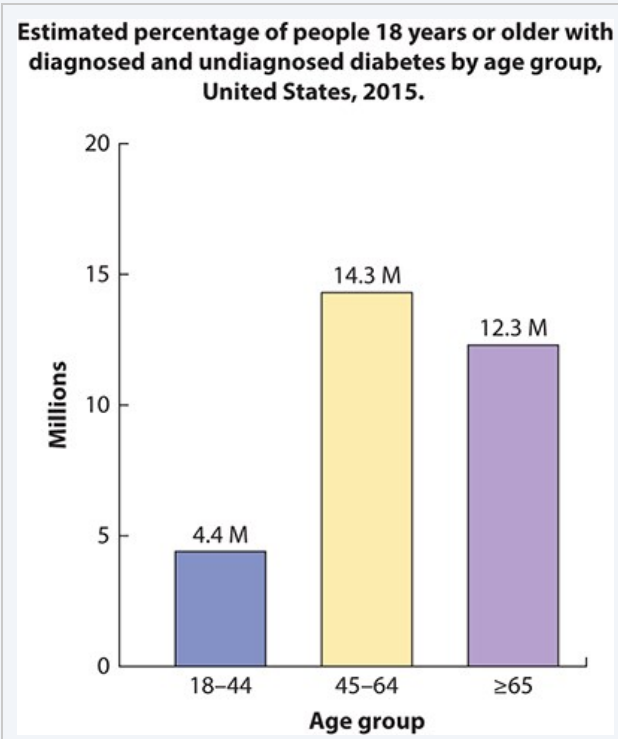
TABLE 7-1

Prevalence of Diabetes in Three Age Groups



Most of the people in the two older age groups with diabetes are being treated for wounds. The startling rise in the number of cases of diabetes, particularly type 2 diabetes, is having a profound effect on the entire health care system. The incidence of diabetes in the United States has doubled in the last 15 years, with the highest prevalence among adults 65–79 years.[5] The national impact will intensify as the population continues to age.

Health care professionals who care for people with chronic wounds who also have diabetes are challenged to achieve wound closure and optimal wound healing outcomes. Diabetes management strategies and wound/foot care are both necessary to provide an environment in which wound closure can occur.

Effects of Diabetes on Wound Healing

Faulty wound healing is a well-recognized complication of diabetes. Current evidence confirms that diabetes inhibits all phases of wound healing via impaired function of the primary cells responsible for wound repair (i.e., neutrophils, macrophages, and fibroblasts), frequently resulting in delayed healing or chronic non-healing wounds. In addition, there is decreased efficacy of cytokines and growth factors in people with diabetes and accompanying hyperglycemia. The accumulation of advanced glycosylation end products (AGEs, also referred to as advanced glycated end products), nitric oxide dysfunction, decreased insulin availability or increased insulin resistance, and altered homocysteine levels also contribute to the complex healing impairments for people with diabetes. Micro- and macrovascular disease, neuropathy, immune dysfunction, and biochemical and hormonal abnormalities all contribute to the altered tissue repair processes in people with diabetes and hyperglycemia (**TABLE 7-2**).[7]

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TABLE 7-2

Mechanisms by Which Hyperglycemia Impairs Wound Healing

• Impaired function of
  ○ Neutrophils
  ○ Macrophages
  ○ Fibroblasts
	• Decreased function of
	  ○ Cytokines
	  ○ Growth factors
		• Accumulation of AGEs
		• Nitric oxide dysfunction
		• Decreased insulin availability
		• Increased insulin resistance
		• Altered homocysteine levels
		• Macrovascular and microvascular impairments
		• Neuropathy
		• Immune function impairments
		• Biochemical and hormone impairments

CASE STUDY

INTRODUCTION

Mr. KM is a 60-year-old male with a 15-year history of type 2 diabetes who presents with a full-thickness neuropathic ulcer on the first metatarsal head of the right foot that has been present for 4 weeks (**FIGURE 7-2**). Past medical history includes hypertension, coronary artery disease, and peripheral arterial disease. His fasting blood glucose (FBG) reported by the patient is usually 120 mg/dL. During the subjective interview, Mr. KM was unable to verbalize the importance of his hemoglobin A1C test results and had no knowledge of what this test meant to the overall management of his diabetes. He was given a diabetes management pamphlet from his physician and had some education from the pharmacist about his diabetes medications. He has had no formal diabetes self-management education. **TABLE 7-3** contains the patient history and laboratory tests that were ordered. **TABLE 7-4** contains the findings from the patient and foot examination.

CASE STUDY PLAN OF CARE

Diabetes Management Strategies Mr. KM has poor blood glucose control as demonstrated by the high hemoglobin A1C of 9.0%, as well as the fasting blood glucose of 395 mg/dL (**TABLE 7-5**). Initial care of this person involves getting the blood glucose under control. He needs to be referred back to his primary care physician for a review of his overall diabetes management strategies. With blood glucose levels this high, the patient probably needs to begin insulin injections in addition to his current or updated oral medications (**TABLE 7-6**). In addition, attention to the patient's lipid panel is needed as his cholesterol and triglycerides are high, which may contribute to coronary and peripheral arterial disease. Overall diabetes self-management education is important for this patient with an emphasis on medical nutrition therapy to assist with nutrition choices that will help manage blood glucose, cholesterol, triglycerides, and weight.

DISCUSSION QUESTIONS

Based on the findings in **TABLE 7-4**, what tests and measures are indicated?

What findings indicate that the patient may have impaired healing potential?

What other medical disciplines would be helpful in treating this patient, other than specific wound care?

FIGURE 7-2

Case study Full-thickness diabetic foot ulcer on the first metatarsal head.

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FIGURE 7-3

A, B. Calcaneal eversion and too many toes The posterior view of the foot illustrates calcaneal eversion resulting in two toes visible on the lateral side, whereas if the ankle is in neutral no toes are visible on the lateral side.

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FIGURE 7-4

Hubscher maneuver

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FIGURE 7-5

Intrinsic muscle wasting with retraction of the digits

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FIGURE 7-6

Silfverskiold test

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FIGURE 7-7

Plantar foot with calluses on first and fifth metatarsal heads

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FIGURE 7-8

Semmes–Weinstein monofilament testing Semmes–Weinstein monofilament testing is used to determine the loss of pressure sensation. The filament is placed on the test spot and pressed until the filament bends. The patient responds as to whether or not the pressure is felt. Inability to feel the 5.07 monofilament (which exerts 10 g of pressure) is termed *loss of protective sensation.*

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TABLE 7-3

Case Study Information

Medical Diagnoses		

• 15-year history of type 2 DM
• Hypertension
• Depression
• Peripheral arterial disease
  ○ ABI = 1.2
  ○ TBI = 0.5
	• Osteoarthritis
	• Nonproliferative retinopathy
	• No known medication or dietary allergies
	• Full-thickness ulceration on the submetatarsal head of the right foot; unable to probe to bone	

Laboratory test results		**Medications/Medical Indication**

• Albumin: 3.9 g/dL (normal)
• Cholesterol: 260 mg/dL (high)
• Triglycerides: 160 mg/dL (high)
• HDL cholesterol: 25 mg/dL (low)
• LDL cholesterol: 130 mg/dL (high)
• Total protein: 6.0 g/dL (normal)
• Fasting glucose: 395 mg/dL (high)
• Hemoglobin HbA1C: 9.0% (high)
• BUN: 16 mg/dL (normal)
• Creatinine: 1.9 mg/dL (high)
	• Complete blood cell count
	• White blood cells: 7.0 × 10^9/L (normal)
	• Red blood cells: 4.0 × 10^{12}/L (normal)
	• Hemoglobin: 12.0 g/dL (normal)
	• Hematocrit: 40% (normal)
	• Neutrophils: 60% (normal)
	  ○ Lymphocytes: 30% (normal)
	  ○ Platelets: 270 × 10^9/L (normal)
		• Glyburide: diabetes
		• Furosemide: CHF
		• Lisinopril: HTN
		• Zoloft: depression
		• Coumadin: anticoagulation
		• Eye drops: glaucoma
		• Ibuprofen: joint pain
		• Neurontin: neuropathic pain

• **Vital Signs: HR: 84, BP: 155/94, Resp.: 16, Temperature: 99.2** • **Pain Assessment—0/10 (peripheral neuropathy present)**		

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TABLE 7-4

Foot Examination for the Case Study

Weight-Bearing Assessment	Non-Weight-Bearing Assessment	
• Stance position—pronated • LMI (lateral malleolar index)—(–)6 mm • Calcaneal eversion—4° everted • Too many toes sign—B/L (**FIGURE 7-3**) • Hubscher maneuver—negative (no arch elevation with dorsiflexion of the hallux indicating hallux limitus) (**FIGURE 7-4**) • Genu valgum absent • Genu recurvatum noted (compensation for equinus) • Contraction of the digits 2–5 noted B/L—minimal digital purchase in stance (**FIGURE 7-5**)	• Silfverskiold test 0° dorsiflexion knee bent and knee straight (**FIGURE 7-6**) • Knee hyperextension noted • Calcaneal inversion 8° • Calcaneal eversion 4° • Midtarsal joint hypermobility noted (high axis orientation as evidenced by transverse plane motion greater than frontal plane motion) • Hallux ROM limited to 30° dorsiflexion (65° needed for unrestricted ambulation) • Hallux interphalangeal joint hypermobile secondary to force transfer to the IPJ because of the hallux limitus • Fixed flexion contracture noted at the IP joints with subluxation and dorsal contracture of the metatarsophalangeal joints 2–4 (inability of either joint to extend and flex to 0°) • Adductovarus fifth toe B/L • Hallux abductovalgus deformity with moderate bunion B/L • Plantar calluses at the hallux IPJ and under the first and fifth met heads B/L (**FIGURE 7-7**) • Dorsal corns (heloma) second and fifth digits B/L	
Vascular Assessment	**Neurologic Assessment**	**Gait Analysis**
• Dorsalis pedis pulse ¼ B/L • Posterior tibial pulse nonpalpable • Hair absent from the toes and dorsal foot • Capillary refill time with feet horizontal 4 seconds (normal ≤2 seconds) • Digital temperature warm • Plantar rubor noted • No ankle or limb edema noted • No varicosity noted • Nails dystrophic with evidence of onychomycosis on all 10 toes	• Loss of protective sensation— LOPS hallux, third and fifth toes, first, third, and fifth metatarsal heads and dorsal first interspace B/L. Able to feel the 5.07 monofilament midfoot and heels B/L (**FIGURE 7-8**) • Pinprick same as above • Light touch absent below the ankle B/L • Position sense absent DIPJs and MPJs B/L but WNL ankle and MTJs • Manuel muscle test WNL with 4+/5-5/5 strength in all muscles tested • Negative Babinski, negative clonus	• Observe barefoot and with shoes • Look for abnormalities at heel strike early and late midstance and propulsion • Observe postural changes during the swing phase • Note step and stride length, cadence, foot placement and balance, apropulsive tendencies • Check for early heel off, midfoot break, and abductory twist at heel off

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TABLE 7-5

Critical Values for Hemoglobin HbA1C and Glucose

HbA1C%	Average Glucose
12.5% 12%	312 mg/dL 298 mg/dL
11.5% 11%	283 mg/dL 269 mg/dL
10.5% 10%	255 mg/dL 240 mg/dL
9.5% 9%	226 mg/dL 212 mg/dL
8.5% 8%	197 mg/dL 183 mg/dL
7.5% 7%	169 mg/dL 154 mg/dL
6.5% 6%	140 mg/dL 125 mg/dL
5.5% 5.0%	111 mg/dL 97 mg/dL

The numbers in green indicate normal values, and as the numbers progress to the top of the chart, they become more critical.

TABLE 7-6

Type 2 Diabetes Medications (Except Insulin)

Class of Medication	Generic Names	Brand Names	Target Organs	Mode of Action
Sulfonylureas	Glyburide Glipizide Glimepiride Micronized glyburide	Diabeta Micronase Glynase Glucotrol Glucotrol XL Amaryl	Pancreatic β cells	Stimulate pancreatic β cells to release insulin. All sulfonylurea drugs have similar effects on blood glucose levels, but differ in side effects, how often they are taken, and interactions with other drugs. This class of drugs is known to cause hypoglycemia.
Biguanides	Metformin	Glucophage Glucophage XR Fortamet	Liver, adipose tissue, skeletal	↓ liver glucose production. ↓ insulin resistance in periphery.

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		Riomet (liquid metformin) Glumetza	muscle	
Meglitinides	Repaglinide	Prandin	Pancreatic β cells	Stimulate β cells to release insulin immediately after eating. Must be taken with meals. Less likely than sulfonylureas to cause low blood glucose.
D-phenylalanine derivatives	Nateglinide	Starlix	Pancreas	Stimulate insulin secretion from the pancreas. Extent of insulin release is glucose dependent and diminishes at low glucose levels, creating less of a risk for hypoglycemia.
Alpha-glucosidase inhibitors	Acarbose Miglitol	Precose Glyset	Small intestine, pancreas	Lower glucose by blocking breakdown of carbohydrates in the intestine, thereby slowing the rise in blood glucose after meals.
Thiazolidinediones	Pioglitazone Rosiglitazone	Actos Avandia	Peripheral tissues, liver	Decrease insulin resistance in the muscle and fat and also reduce glucose production in the liver. Make body more sensitive to the effects of insulin.
DPP-4 inhibitors	Sitagliptin Saxagliptin Linagliptin	Januvia Onglyza Tradjenta	Pancreas Muscles Liver	Inhibit DPP-4, thereby increasing insulin synthesis/release from pancreatic β cells. Can indirectly result in increased glucose uptake in peripheral tissues. Decrease liver glucose production. Lower glucose only when they are elevated.
Glucagon-like peptide-1 analog	Exenatide Liraglutide	Byetta Victoza	Stomach, liver, pancreas, brain	Enhances glucose-dependent insulin secretion by the pancreatic β cell, suppresses inappropriately elevated glucagon secretion, and slows gastric emptying, subtle effect to reduce appetite. Mechanism of action is not fully understood and still under study. Must be administered by injection.
Bile Acid Sequestrants	Colesevelam	Welchol	Bile acid-binding in liver	Unknown MoA for lowering blood glucose.
COMBINATION ORAL MEDICATIONS These medications combine the actions of two different medication classes and are thought to increase adherence to medication regimes with simpler dosing.				
• Pioglitazone and metformin • Glyburide and metformin • Glipizide and metformin • Sitagliptin and metformin	• Actoplus Met • Glucovance • Metaglip • Janumet • Kombiglyze • Duetact • Prandimet			

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• Saxagliptin and metformin • Pioglitazone and glimepiride • Repaglinide and metformin					

INSULINS FOR TYPE 1 AND TYPE 2 DIABETES

Type of Insulin	Brand Name	Generic Name	Onset	Peak	Duration
Rapid acting	NovoLog Apidra Humalog	Insulin Aspart Insulin Glulisine Insulin Lispro	15 minutes 15 minutes 15 minutes	30–90 minutes 30–90 minutes 30–90 minutes	3–5 hours 3–5 hours 3–5 hours
Short acting	Humulin R Novolin Actrapid	Regular (R)	30–60 minutes	2–4 hours	5–8 hours
Intermediate acting	Humulin N Novolin N	NPH (N)	1–3 hours	4–12 hours	12–18 hours
Long acting (basal or background insulins)	Levemir Lantus Toujeo Tresiba Basaglar	Insulin detemir Insulin glargine Insulin glargine Insulin degludec Insulin glargine	1 hour	Peakless	20–26 hours
Premixed NPH (intermediate acting) and regular (short acting)	Humulin 70/30 Novolin 70/30 Humulin 50/50	70% NPH and 30% regular 50% NPH and 50% regular	30–60 minutes 30–60 minutes	Varies Varies	10–16 hours 10–16 hours
Premixed insulin lispro protamine suspension (intermediate acting) and insulin lispro (rapid acting)	Humalog mix 75/25 Humalog mix 50/50	75% insulin lispro protamine and 25% insulin lispro	10–15 minutes 10–15 minutes	Varies Varies	10–16 hours 10–16 hours

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		50% insulin lispro protamine and 50% insulin lispro			
Premixed insulin aspart protamine suspension (intermediate acting) and insulin as part (rapid acting)	NovoLog mix 70/30	70% insulin aspart protamine and 30% insulin aspart	5–15 minutes	Varies	10–16 hours

From National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), National Institutes of Health (NIH); Joslin Diabetes Center; Medication Package Inserts.

One example of a diabetes-mediated impairment in wound healing is susceptibility to infection. Under normal conditions, during the hemostasis healing phase there is immediate fibrin plug formation as platelets aggregate at the wound site. The platelets release various growth factors and cytokines, which then recruit inflammatory cells; however, in a hyperglycemic environment, there is a delay in fibrin plug formation, leaving the wound open to contaminants. In addition, there is a delay (or decrease) in the release of growth factors and cytokines, causing impaired recruitment of the inflammatory cells. With this delay, the individual is at risk for infection. In fact, people with diabetes have more frequent infections than patients without diabetes.[7] On the other side, infection will increase the stress-related hormones needed to fight illness, which in turn increases the blood glucose levels. Therefore, high glucose levels in an individual whose blood sugars are usually well controlled may be a sign of localized infection.

CLINICAL CONSIDERATION

The hemoglobin A1C test provides a measure of overall blood glucose for the previous 2–3 months. **TABLE 7-5** depicts A1C measurements and how they correlate to blood glucose levels. Note normal and high ranges for A1C. This test is very important to help clinicians and patients understand how the diabetes management plan is working for the individual.[8]

Research in human and animal models has identified many of the changes that contribute to delayed wound healing at the molecular level; however, more research is needed to completely understand how diabetes contributes to faulty tissue repair.[8]

Extensive research has focused on the causes of and interventions for diabetic (also referred to as neuropathic) foot wounds; however, the impairments related to diabetes are much more far-reaching than just the foot, affecting healing of all types of wounds regardless of etiology (eg, pressure ulcers, vascular ulcers, and surgical wounds). In summary, diabetes profoundly impairs the four overlapping phases of wound healing. The underlying mechanisms of the effects of diabetes on wound healing have been extensively investigated over the past few decades; however, complete understanding of the complex and multifaceted pathophysiologic relationship between DM and defective healing continues to elude the scientific community.

CLINICAL CONSIDERATION

People with diabetes are at higher risk for infection because of delays in cellular function at both the hemostasis and inflammatory phases of healing.

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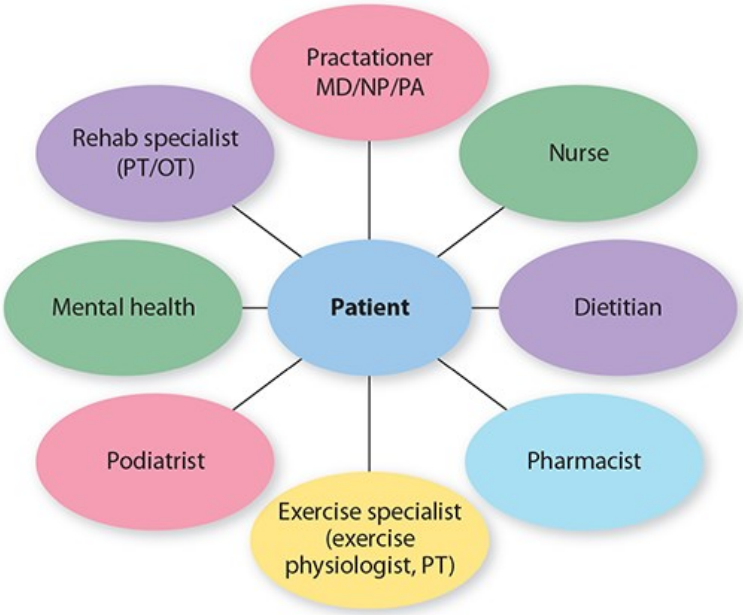
Medical Management and Team Care of Diabetes

Diabetes care is complex and multifaceted, requiring a team approach to patient-centered care, with the patient being an integral member of the team (**FIGURE 7-9**). While the medical team leader is the physician or advanced practice nurse (who uses input, education services, and treatments from other health care providers), the management that occurs on a daily basis is provided by the patient (or by caregivers when the patient is too impaired to perform adequate self-management).[8] The ability of the patient to adequately manage the diabetes depends on extensive and ongoing training, education, and feedback from the health care team for reaching individualized goals for each patient.

FIGURE 7-9

The diabetes care team The team that cares for the patient with diabetes includes these health care specialists, each with an important role in caring for the total patient, with the patient being at the center of the team. The team must have the patient take responsibility for the overall day-to-day management of the disease.



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Effective *wound care* includes a review of the patient's diabetes management strategies. *Without good glucose control, the patient will have difficulty reaching or sustaining wound closure.*

The American Association of Diabetes Educators (AADE) is a multidisciplinary professional organization that empowers diabetes educators as well as other team members responsible for ensuring patients have the knowledge they need to self-manage their disease. Without adequate diabetes self-management education, patients are lost as to how to change their lifestyle and habits to ensure acute and chronic complications are delayed or averted (**TABLE 7-7**). The following list provides the basic elements of a well-rounded diabetes management education program for patients with diabetes, consisting of seven core self-care behaviors that patients should learn in order to delay or avoid complications from diabetes[9]:

- **Healthy Eating**: Patients are taught which foods to eat in a manner that assists with blood glucose control. The different types of macronutrients are described including carbohydrates, protein, and fats. Many people with diabetes think the diet issue is related to ingesting sugar, not understanding that every type of carbohydrate influences blood glucose and that not all carbohydrates are created equal, meaning some cause the glucose to go high, while other have less effects on the overall blood glucose levels. Diabetes educators teach the person how to count carbohydrates, read food labels, measure each serving, develop an eating plan, prevent high or low blood glucose levels, and set goals for healthy eating.[9]
- **Being Active:** Being active is part of being healthy in general and is a necessary component for optimal health when a person has diabetes.

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Physical activity (including exercise) is a powerful modality for the person with diabetes and must be coordinated with the individual's medication and nutrition regimes. The effects of physical activity include lowering blood glucose, lowering cholesterol, improving blood pressure, lowering stress and anxiety, and improving mood. Depending on the associated complications (eg, diabetic retinopathy, diabetic neuropathy, and diabetic nephropathy), certain precautions and contraindications may be implemented to avoid physical activity-induced damage to already-compromised tissues and organs. For example, patients who have severe foot deformities or a history of diabetic foot ulcers need to be instructed in an activity program that does not cause friction and shear to the plantar foot structures. A referral to a physical therapist knowledgeable about exercises and diabetes is recommended for establishing an appropriate exercise program for the patient with diabetes. Individuals will adhere to activity programs more readily when doing activities they enjoy such as dancing, bike riding, or walking the dog. The activity plan must be customized and embraced by the patient for long-term adherence to take place.[9]

CLINICAL CONSIDERATION

Exercises in a standing position that result in the foot being planted while the body moves (eg, squats, the elliptical, rowing) will produce shear between the subcutaneous tissue and the bony prominences of the plantar foot. Bicycling or using an upper extremity cycle ergometer is advised for cardiopulmonary conditioning if the individual has any risk factors for (or a history of) foot ulceration.

- **Glucose Monitoring:** Glucose monitoring helps patients determine the day-to-day control of their disease. Monitoring gives patients a tool to determine objectively if their blood glucose is too high or too low. This information helps them with self-management decisions on a day-to-day basis related to food choices and amounts and types of activities they can do, and helps guide the patients regarding when and whether to see a medical professional. The patient who monitors consistently will discover abnormal glucose excursions and can get help as soon as possible to remedy the higher glucose levels. When monitoring blood glucose, the patient will need the following basic supplies:
  - Lancet—thin needle to prick skin for small sample of blood;
  - Test strips—small pieces of specially prepared paper that absorbs the blood from the lancet prick;
  - Meter (aka glucometer)—small electronic device that reads the test strip and gives glucose readings;
  - Log book—to record the numbers from the blood glucose meter and to share with the health care team managing the diabetes.[9]
- **Medications:** The drug armamentarium for glycemic control for people with diabetes is large and growing as the impairments related to diabetes are better understood. The different drugs and combination therapies address different pathophysiological mechanisms and different organs involved in blood glucose control. The management of T1DM and T2DM is completely different, as these are different diseases with the similar outcome of hyperglycemia.[8] Medications can be oral and/or injectable for T2DM; however, T1DM requires injectable insulin. Inhaled insulin, a new delivery method for insulin, was approved by the FDA in June 2014 for use in both T1DM and T2DM. Inhaled insulin may take the place of short-acting insulins, but do not replace the intermediate and long-acting forms of insulin.[9] See **TABLE 7-6**.
- **Problem Solving:** Preparation and planning are critical components of a well-designed diabetes management program. Patients should monitor their blood glucose levels, looking for too high (hyperglycemia) and too low (hypoglycemia) numbers. However, even the patient who is trying the best to plan well for managing the disease will sometimes find it difficult. Life has all kinds of ups and downs, and no matter how well patients plan, blood glucose can and will go in the wrong direction at some point. An example would be the patient whose blood glucose has been running 180–230 mg/dL for several days, when the random glucose readings are usually between 125 and 145 mg/dL. The patient knows these are higher than normal readings, but may not know why. This person needs professional help to determine if the diabetes management program is still working. There are several reasons the readings may be increasing, eg, illness, a wound infection (if a wound is present), failing beta cells in the pancreas requiring adding insulin to the medication regime, or the glucose strips are out of date. On the other hand, blood glucose can go low, particularly when patients are on oral or injectable diabetes medications. Examples of situations that cause glucose to go low is lack of sufficient food when diabetes medications are taken, too strenuous of an exercise or activity session, or the most common cause—diabetes medications. A bit of health care detective work is necessary when situations such as these arise.[9]
- **Reducing Risks:** Patients who take responsibility for their day-to-day diabetes management can prevent or delay the devastating complications of this disease. Some of the major complications include heart disease, stroke, nephropathy (kidney disease), retinopathy (eye disease), neuropathy, foot abnormalities, and non-healing wounds. In addition to controlling their blood glucose levels, patients with diabetes should

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reduce other risk factors that work in tandem with diabetes and cause the complications to be more severe. Healthy habits to help prevent complications from diabetes include not smoking, seeing the health care practitioner who helps manage the diabetes on a regular basis, annual eye exams, dental care, foot screens, and proper care of the feet and nails.[9]

- **Healthy Coping:** Increased stress in one's life increases blood glucose levels. There are the daily types of stress (eg, traffic and getting the kids off to school), and the more serious issues such as divorce or money problems. Adding life stresses to the strain that the disease state of diabetes causes can be almost overwhelming for some patients. When there are high levels of stress in the diabetes patient's life, it is important that the patient turns to healthy coping methods rather than harmful habits such as smoking, overeating, and excessive alcohol consumption. Some of the recommendations for handling stress for people with diabetes include the following: having a support system with people the patient trusts, physical activity which increases brain endorphins and in turn softens the stress response, thinking positive, and being good to one's self. Sometimes the person is in depression, a common issue for people with diabetes. Some of the signs that a patient with diabetes is depressed include lack of interest or pleasure in personal or social activities, not wanting to discuss their diabetes issues with family or friends, sleeping for long periods each day, and not providing daily self-care from a personal and/or diabetes perspective. Some people get so depressed they decide to give up on themselves and stop with their good diabetes management. Recognizing patient depression is a health care assessment activity and should be done for all people with diabetes, both those with and without complications from their disease.[9]

TABLE 7-7

Diabetes Education Content Areas[9]

- Describe the *DM disease process* and *treatment options.*
- Incorporate *healthy eating* into lifestyle.
- Incorporate *physical activity* into lifestyle.
- Use *medication(s) and devices safely* and for maximum therapeutic effectiveness.
- *Monitor blood glucose* and other parameters; interpret and use the results for self-management decision making.
- Prevent, detect, and treat *acute complications* (ie, hyperglycemia and hypoglycemia).
- Prevent, detect, and treat *chronic complications* (eg, heart and kidney disease, retinopathy, Charcot foot disease).
- Healthy coping to address psychosocial issues and concerns.
- Develop personal strategies (problem solving) to promote health and behavior change.

Challenges in Caring for the Patient with Diabetes

Education

There are many challenges in providing care for individuals with diabetes. Education is required not only for self-management of diabetes, but also for self-management of the wound. Many people with chronic wounds and diabetes do not have the necessary information to adequately manage the disease, especially in the presence of a chronic wound that causes both psychological and physiological stress. The standard of care for individuals with diabetes is referral to a comprehensive diabetes education program upon diagnosis of the disease[8]; however, many do not have access to a diabetes education center. Such individuals thus present to the wound care clinic with a critical deficiency in their ability to manage the disease. This deficiency itself could be a contributing factor to both the development of the wound and the impaired wound healing.

CLINICAL CONSIDERATION

The wound care clinician *must* review the patient's blood glucose values (both the capillary blood glucose and the hemoglobin A1C) to determine if the patient needs to see a physician to adjust the medications in order to control blood glucose.

Depression and Burnout

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As mentioned previously many people with diabetes have varying levels of depression,[8] which can mildly or severely compromise self-management, depending on their coping abilities. If the individual's diabetes management strategies are compromised by depression, the provider can expect more challenges for wound healing. Diabetes alone conveys psychological, social, and financial burdens on the affected individual; a wound creates additional psychological, social, and financial burdens.

CLINICAL CONSIDERATION

Many people with diabetes, especially type 2, do *not* receive adequate education for successful self—management, yet they are blamed for being non-adherent to their diabetes management program. Also, almost everyone backslides at times—some more frequently than others—on a diabetes management program. However, such lapses can simply represent the understandable difficulty of completely changing and constantly monitoring major aspects of one's daily life, including nutrition, physical activity, and often complex medication regimes. Thus, people with diabetes need both extensive initial education and ongoing support and encouragement from people familiar with the management strategies of this disease, including wound care professionals.

Burnout is a potential problem for everyone concerned—the patient can become burned out from living with diabetes and its associated complications continuously, with no respite, and the wound care team can become burned out by the impact of diabetes on wound healing and the perception that the patient is not optimally self-managing the disease or the wound, thereby "sabotaging" the wound care plan. The psychosocial impact of diabetes is life altering, especially with the addition of chronic complications that frequently accompany diabetes, including unsightly, odiferous, and difficult-to-manage wounds. There are no easy answers or rote formulas for these challenges; providers must do their best to provide comprehensive support to these patients and their families.[8]

Adherence

The patient's adherence to the overall disease management plan is critical. However, before placing the term *non-compliant* or *non-adherent* on the person with diabetes, the wound care clinician should assess whether the patient has the best diabetes management plan individualized to his or her needs. The clinician must assess the patient's ability to self-manage the diabetes and provide an appropriate referral as needed to address deficiencies where they exist. The status of the patient's diabetes control is prerequisite knowledge in order for the wound care clinician to help the patient create an environment for wound closure and healing. **TABLE 7-8** lists helpful strategies for the clinician who is working with a patient who is having difficulty with adherence.

TABLE 7-8

Strategies to Help a Patient Develop Adherence to a Comprehensive Diabetes Management Program

1. Provide a referral for diabetes self-management education, if necessary.
2. Provide ongoing monitoring and encouragement.
3. Emphasize that DM self-management is a lifelong process.
4. Help the patient understand the health benefits, including wound healing, that make his or her hardwork worthwhile.
5. Address relapse management by expressing understanding of the difficulty of adhering to the diabetes management program day in and day out.
6. Help the person realize that one instance of nonadherence should be seen in the context of just one lapse and not taken as proof that he or she is unable to be successful. Such an approach promotes rapport and sends the message that the clinician and the patient are on the same team.

The Wound Care Clinician's Role

The initial examination of the patient with a wound includes a basic assessment of the patient's diabetes management plan to ensure that the disease is under control. **TABLE 7-9** is a suggested list of intake components to consider, and **TABLE 7-10** lists the diagnostic criteria for diabetes.

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TABLE 7-9

Important Questions to Ask a Person with Diabetes When Performing the Wound Evaluation

- Have you had a series of diabetes self-management classes?
  - Yes: How long ago? _____ months _____ years
- What medications do you take for DM? List the names and dose of each medication.
- How often do you monitor your blood glucose? What were they this morning when you awakened?
- Do you take your diabetes medications regularly without fail?
  - Yes
  - No: How often do you take your medications?

TABLE 7-10

Type 2 Diabetes Diagnostic Criteria for Adults

	TESTS		
Stage	**Fasting Plasma Glucose[a] Test**	**Casual Glucose[b] Test[c]**	**Oral Glucose Tolerance Test[d]**
Normal	<100 mg/dL	__	2-hour PPG <140 mg/dL
Prediabetes (increased risk for diabetes; also known as impaired glucose tolerance)	100–125 mg/dL	A1C = 5.7–6.4%	2-hour PPG 140–199 mg/dL
Diabetes	≥126 mg/dL	≥200 mg/dL (plus signs and symptoms)	2-hour PPG ≥200 mg/dL
		A1C ≥6.5%	

Abbreviation: PPG, postprandial glucose.

[a]Fasting means no calorie intake for 8 hours.

[b]Casual means testing any time of day without regard to time since last meal; signs and symptoms are the classic ones of polyuria, polydipsia, and unexplained weight loss.

[c]The A1C should be performed in a laboratory using a method that is NGSP certified and standardized to the Diabetes Control and Complications Trial assay.

[d]2-hour PPG indicates 2-hour postload glucose.

Data from American Diabetes Association. Standards of medical care in diabetes. *Diabetes Care.* 2018; 41(suppl 1):S1–S2.

A typical case scenario may present as follows: a patient with diabetes presents with a wound with signs and symptoms of infection. The blood glucose level is 345 mg/dL. The clinician needs to determine which came first: chronic hyperglycemia, which made the patient more susceptible to infection, or the infection, which caused the blood glucose levels to increase. A comparison of the capillary blood glucose levels with a current hemoglobin A1C test can help make this determination. If the A1C value is high, then the blood glucose has been out of control for at least several months and diabetes management strategies are of paramount importance. If the A1C shows adequate control, then the diabetes has been well managed, and the infection has caused the elevated blood glucose levels. At this point, the patient may require the addition of insulin to the diabetes medication regime—at least

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temporarily—to help control the infection-instigated elevated blood glucose. The wound care clinician can make this recommendation to the provider who manages the patient's diabetes. Thus, object glycemic tests can be used to help manage wound healing, remembering that diabetes is ever present and must be managed at all times to help create the best wound closure and healing outcomes.

CLINICAL CONSIDERATION

Often a person with type 2 diabetes is further along in the disease process of β-cell failure than when he or she first started treatment. Because type 2 is a progressive disease, the oral medications may no longer provide the control which they once did; that is, the β cells are not able to respond to the insulin-secreting mechanism of the medications, or the patient's insulin resistance has increased and the medications that address insulin resistance are not able to function well enough to lower the blood glucose. The wound care clinician may be the health care provider who recognizes or suspects a diabetes treatment regime needs updating to provide optimal blood glucose levels.

CLINICAL CONSIDERATION

Sometimes people who are on fixed or low incomes take their medications sporadically to try to make them last longer. Another scenario is when an older person is confused about the medication regime altogether and embarrassed to tell someone that he or she needs help with a functional method to adhere to the medication plan of care. Also, the wound care clinician may be the first to recognize recent mental status changes or abuse and neglect issues that are impacting the patient's diabetes self-management. Again, adherence or non-adherence is often a complex issue reflecting other, underlying areas of concern.

Conclusion (Diabetes)

The wound care team needs a working knowledge of the diagnosis of diabetes, diabetes management strategies (including the most commonly associated complications and the effects of diabetes on wound healing), as well as proficiency in performing a thorough assessment of the patient and the wound. While it is not the responsibility of the wound care team to manage the person's diabetes—that duty belongs to the patient and the practitioner responsible for the medical management—the wound care team should assess if the disease is being managed as successfully as possible and recognize when other diabetes interventions and counseling need to be considered for optimal wound closure and healing outcomes.[10,11]

CLINICAL CONSIDERATION

The wound care clinician *must* review the patient's blood glucose values, both the capillary blood glucose and the hemoglobin A1C, to determine if the patient needs to see the primary care physician or endocrinologist to adjust the diabetes medications.

THE DIABETIC FOOT

The diabetic foot is a complicated pathological entity suffering from varying degrees of physiological and biomechanical balance and imbalance. Prolonged exposure to slowly elevating blood glucose leads to glycosylation of multiple organ systems and progressively advancing neurovascular crises triggered by the loss of system homeostasis. The “perfect storm” of intrinsic and extrinsic stressors associated with diabetes causes a system collapse that results in a breakdown of the foot's ability to manage the combination of individual stressors, thereby leading to a breakdown sufficient to cause ulceration and/or neuroarthropathy of the diabetic foot.[12]

Once the skin has broken and exposed the underlying tissues to the risk of infection, the patient and the practitioner have a relatively short period of time within which to address the component problems and restore balance to the system, otherwise complications such as osteomyelitis or amputation may ensue. Carlyle Begay, in a presentation delivered to the National Indian Health Bureau in January 2012, referred to this time period as “the golden hour for the diabetic foot,” a concept coined from the emergency management of cardiovascular and cerebrovascular accidents (**FIGURE 7-10**).[13]

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FIGURE 7-10

Bilateral submetatarsal two ulcers secondary to a short first metatarsal The bilateral plantar wounds on the second metatarsals are a result of a short first metatarsal. These wounds would have a relatively short period of time for treatment before the risk of infection or osteomyelitis is greatly increased.

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From the onset of the wound, there are approximately 30 days or 4 weeks to restore homeostasis and thereby prevent further tissue breakdown, infection, and progression to amputation. Standard of care requires the introduction of definitive care before 4 weeks; advanced therapies to heal the wound can be initiated at that time if 50% wound closure has not occurred. Dr Peter Sheehan conducted a large, prospective, multicenter trial of 203 diabetic patients that assessed the ability of the *4-week healing rate* to predict complete healing at 12 weeks. He concluded that "patients in whom ulcer size fails to reduce by half over the first 4 weeks of treatment are unlikely to achieve wound healing over a reasonable period."[14]

There is a fivefold increase in the risk of infection in wounds that progress beyond 4 weeks of therapy and a 155 times greater risk of amputation once an infection develops.[15]

A panel created for the American Diabetes Association Consensus Development Conference on Diabetic Foot Wound Care in April 1999 concluded, "Any wound that remains unhealed after 4 weeks is cause for concern, as it is associated with worse outcomes, including amputations."[16] It is evident from these studies that a wound on a diabetic foot is a very serious event and must be treated as such from day one. Early interventions such as risk assessment, educational initiatives, and aggressive treatment interventions can literally be life saving for many patients with diabetes.

Diabetes and the Effects on the Systems of the Foot

Foot problems are common in patients with diabetes and 25% of them, or approximately 6.8 million people, will develop a foot ulcer during their lifetime.[17,18] Once an ulcer has developed, the 5-year survival rate for a patient with diabetes is approximately 45%, a figure that is lower than that for both prostate and breast cancer. Despite the dangers and the risks associated with ulceration in the diabetic population, the problem has received very little press in comparison to other more politically interesting diseases.[19] For many years, foot complications have been the leading cause of hospitalization for patients with diabetes.[20] Almost 30% of diabetics aged 40 years or older have impaired sensation in the feet (ie, at least one area that lacks feeling), or approximately 8 million individuals (60–70% of people with diabetes) have mild-to-severe forms of nervous system damage affecting both sensory and motor systems.[21] The major system failure in diabetics leading to the development of an ulcer is the *loss of protective sensation* in the lower extremity. Combined with intrinsic muscle loss and extrinsic muscle imbalance, this leads to increasing biomechanical stress in areas of high pressure and eventual tissue loss. The skin breakdown is a combination of pressure ischemia, reperfusion injury, expanding sublesional microhemorrhage and micronecrosis, and shearing injury, all of which lead to an accumulation of coalescing blood and transudate within or beneath the skin. The skin eventually ruptures, opening the subcutaneous tissues to bacterial invasion from the skin surface (**FIGURES 7-11** and **7-12**).

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CLINICAL CONSIDERATION

After a thorough history, the physical examination assesses the dermatological, musculoskeletal, neurological, and vascular systems of the lower extremity. Skin condition and lesions are noted and recorded in addition to the measurements of the wound or wounds on the foot.

FIGURE 7-11

Charcot foot with dissecting sublesional hematoma

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FIGURE 7-12

Deroofed hematoma with exposed full-thickness ulcer

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Diabetic dyshidrosis or dry skin secondary to the loss of normal skin perspiring (a result of autonomic nervous dysfunction) is documented, and

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patient education about the daily use of an emollient is included in the care plan, along with consultation with a podiatric or allopathic physician for a prescription hydrating cream (**FIGURE 7-13**). Neurologic assessment includes the Semmes–Weinstein monofilament test, reflexes, biothesiometer testing, muscle strength and function, visual gait and posture analysis, and some quantification of plantar pressures (**FIGURE 7-8**). **FIGURE 7-14** illustrates the 10 sites that are tested with the monofilaments. The PressureStat device is an excellent tool for obtaining a quick quantifiable assessment of plantar pressures (**FIGURE 7-15**).

CLINICAL CONSIDERATION

An ankle-brachial index above 1.3 is considered unreliable for a patient with diabetes due to calcification of the arteries, in which case a toe-brachial index would be performed instead.

FIGURE 7-13

Severe dyshidrosis before and after emollient

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FIGURE 7-14

Diagram of sites tested with the Semmes–Weinstein monofilaments Ten sites are tested on each foot to determine the loss of pressure sensation. This is considered one of the most reliable risk factors for diabetic foot ulceration.

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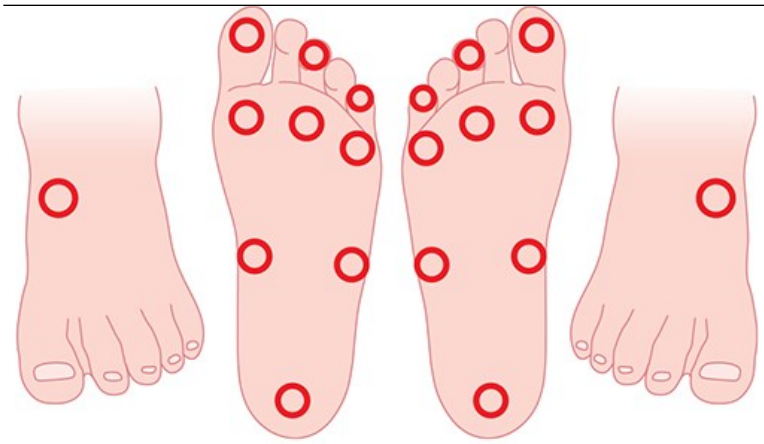

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FIGURE 7-15

PressureStat pressure mat The pressure mat is used to obtain a quantifiable estimate of dynamic plantar foot pressures.

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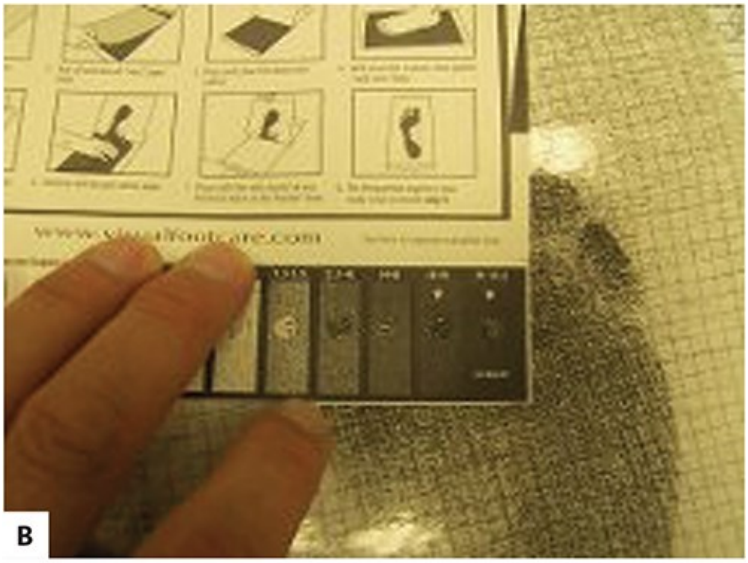

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The major risks for ulceration are determined by the neurologic and musculoskeletal findings, and the major risk for amputation is determined by the vascular findings. A complete vascular screening includes pulses, trophic skin changes (eg, hair loss, skin thinning, nail thickening, and pigment changes), capillary refill time, and the calculation of both the ankle brachial index (ABI) and the toe-brachial index (TBI). ABIs are often falsely elevated due to calcification of the arteries, leading to artificially elevated vessel occlusion pressures. The toe arteries are much less likely to become calcified,

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thereby providing a more accurate impression of the true tissue perfusion. Near infrared spectroscopy (NIRS) is another way to measure skin perfusion. The hand-held device measures reflected light from the blood cells to determine a ratio of oxygenated to deoxygenated hemoglobin, thereby accurately measuring both the amount of blood reaching the tissue and the level of oxygenation of that blood.[22] One study suggests that wounds should have at least 40% oxygenated to deoxygenated hemoglobin in order to achieve wound healing.[23] (Refer to Chapter 4, Vascular Wounds, for more information on vascular testing.)

The musculoskeletal assessment of the foot is performed in both weight-bearing and non-weight-bearing positions. Deformities and their severity are assessed along with any motion restrictions, especially restriction produced by the presence of equinus. A simple classification system was developed at Temple University to assess the depth of wounds and the degree of foot deformity in order to determine the type of off-loading device necessary to heal the wound (**TABLE 7-11**).

TABLE 7-11

Temple University Classification for Footwear Selection[a]

	Depth of Ulcer	Depth of Ulcer	Depth of Ulcer
	1	2	3
	Pre- or Postulcerative Lesion	Superficial Not Involving	Deep Involving Tendon
Degree of Deformity	**Fully Epithelialized**	**Tendon Capsule or Bone**	**Capsule or Bone**
A Mild (normal appearance to minimal abnormality noted)	Standard depth footwear Simple multilaminate insoles Accommodative padding Digital orthoses	Removable cast walker Football or felted foam dressing Surgical shoe with total contact molded orthosis PWB with ambulatory aid	iTCC Football dressing Felted foam TCC PTB/hawk/CROW NWB with ambulatory aid
B Moderate (moderately odd-looking foot alignment—single focal osseous or joint deformity)	Depth footwear Custom orthotic inserts Digital orthoses	iTCC/TCC Removable cast walker Football dressing Felted foam PWB with ambulatory aid	iTCC Football dressing Felted foam TCC PTB/hawk/CROW NWB with ambulatory aid bedrest
C Severe (very odd-looking foot with multiple osseous or joint deformities)	Custom depth or molded shoes Custom orthotic inserts Custom molded ankle Foot orthosis (torch/CROW/PTB Arizona/platinum orthoses)	TCC/ iTCC Football dressing or felted foam in an RCW PTB/hawk/CROW/torch NWB with ambulatory aid	TCC/iTCC Football dressing or felted foam in an RCW NWB with ambulatory aid bedrest

[a]All shoes and fixed ankle devices have rocker soles to allow for a smooth gait and reduction of forefoot loading during ambulation.

Pictorial Glossary of Transitional Off-Loading Devices

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See FIGURES 7-16 to 7-29.

FIGURE 7-16

Total contact cast (TCC)

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FIGURE 7-17

A, B. Instant total contact cast (iTCC) with ties to ensure compliance

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FIGURE 7-18

Removable cast walkers (RCWs) with insole

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FIGURE 7-19

The Rader football dressing

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FIGURE 7-20

The felted foam dressing

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FIGURE 7-21

The D'Arco wound healing shoe D'Arco wound healing shoe consists of an open toe shoe with a rocker bottom and four layers of dense foam. Elliptical cut-outs are made in the middle two layers of foam to relieve the pressure on and around the plantar DFU.

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FIGURE 7-22

Patellar tendon bearing ankle foot orthoses with rocker soled depth shoe Patellar tendon bearing (PTB) with ankle foot orthosis (AFO) can be combined with a rocker soled depth shoe, or in this case, with an extra wide shoe with molded insert. The toe deformity would determine if the athletic shoe is sufficient to prevent dorsal toe friction.

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FIGURE 7-23

Limb-load removable cast walker

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FIGURE 7-24

Charcot restraint orthopedic walker or CROW

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FIGURE 7-25

Modified Carville healing sandal—surgical shoe with a total contact heat-molded insert

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FIGURE 7-26

Commercial depth shoe with rocker sole and total contact insert

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FIGURE 7-27

Carville 22° rocker sole

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FIGURE 7-28

Custom molded shoe

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FIGURE 7-29

Silicone molded digital orthosis

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Eighty-five percent of all lower limb amputations in patients with diabetes are preceded by a foot ulcer.[17,24] Those who develop a foot ulcer have a 55 times greater risk of infection[23] and if a DFU is open for 30 days or longer that risk quadruples.[15] The cost to the health care system is astronomical. Using conventional care, the average cost to obtain a healed ulcer is $56,000, due in part to the relative inefficiency of what is considered good wound care.[25]

In a recent meta-analysis, Margolis concluded that after 12 weeks of good wound care only 24.2% of ulcers were healed, and after an additional 8 weeks of the same care or 20 total weeks, only 30.9% of those same wounds would have achieved complete closure.[26]

The total annual cost of diabetes treatment in 2002 (including direct and indirect costs) was estimated at $132 billion, or 1 out of every 10 health care dollars spent in the United States.[27] A patient hospitalized for a DFU can expect a 59% longer length of stay than a diabetic patient hospitalized without a foot ulcer.[28] A lower-extremity amputation (LEA) will be required by 14–24% of those DFU patients[20] and unfortunately the incidence of LEA in people with diabetes continues to rise despite deliberate efforts to prevent amputations in the last decade.[16]

Biomechanical Changes with Diabetes

Diabetes has a particularly devastating effect on the form and function of the human foot. The human foot is an intricate ground interface for the human body providing sensory input, balance, and stability. The 26 bones, ligaments, and muscles are organized into a highly efficient mechanical system for maintaining balance and posture, sensing ground position and surface characteristics, and converting muscular and ground reactive forces into the various movements required to stand and move. The many nerves of the plantar surface provide constant feedback to the spinal cord and brain where an elaborate system of reflex and intentional movements keep the body's center of gravity (COG) balanced between the two feet in the bipedal stance or over the weight-bearing foot in unilateral single support.

Gait is the intentional transportation of the body's COG in a forward manner. Major forces are generated in the sagittal plane with balance and control forces modulated in the frontal and transverse planes. The muscles of the lower extremity work together in an attempt to maintain the COG either between the two legs or directly over the plantigrade foot (**FIGURES 7-30** and **7-31**).

FIGURE 7-30

Frontal plane view of the transmission of forces through the center of gravity in each foot

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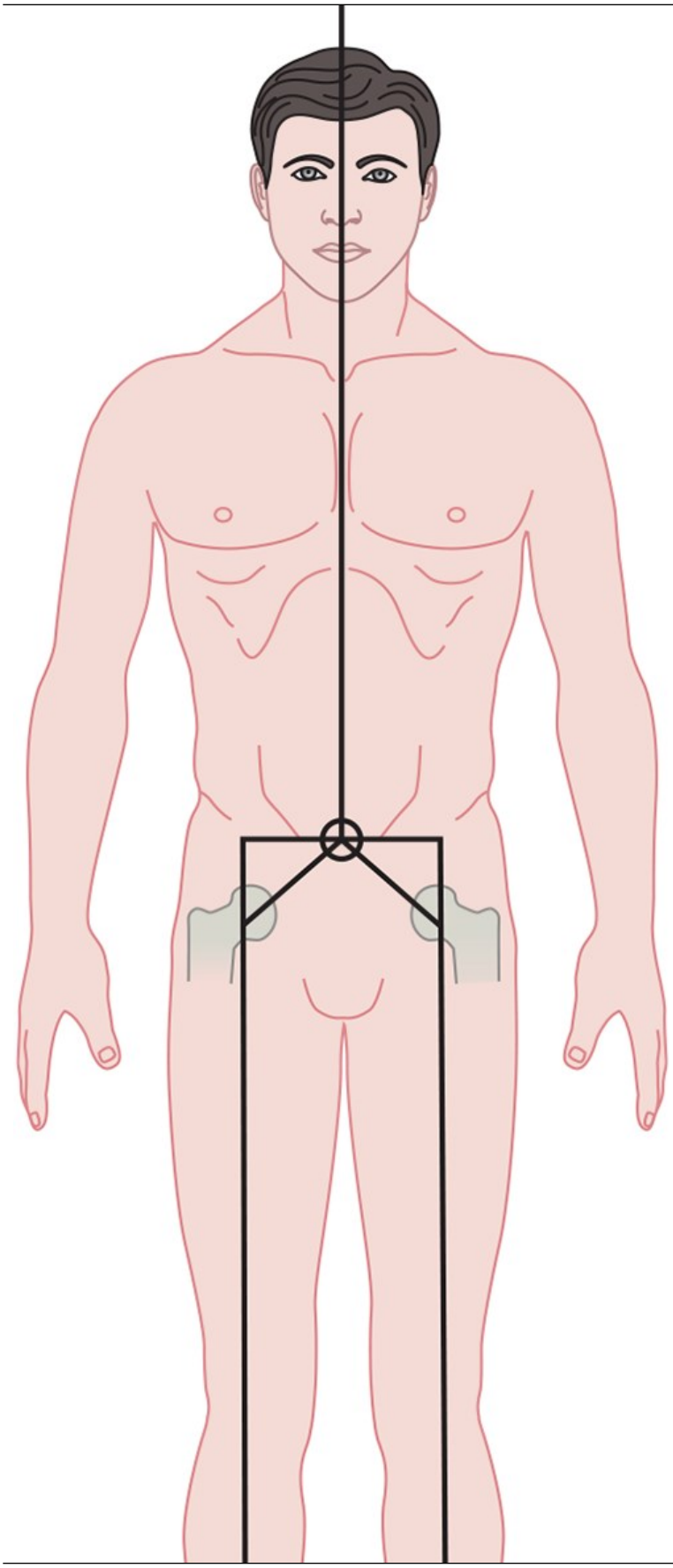

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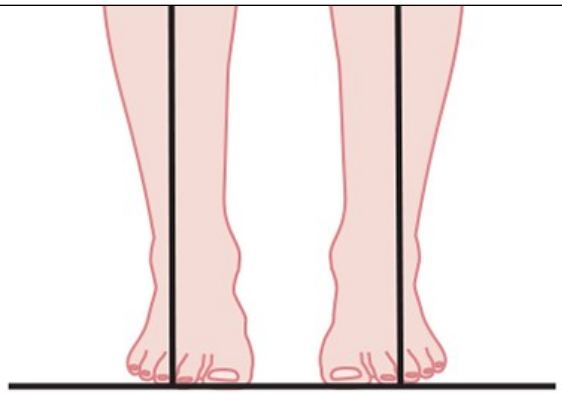

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FIGURE 7-31

Close-up of the frontal plane forces delivered through the foot center axis of support (Used with permission from Dr. Alan Whitney.)

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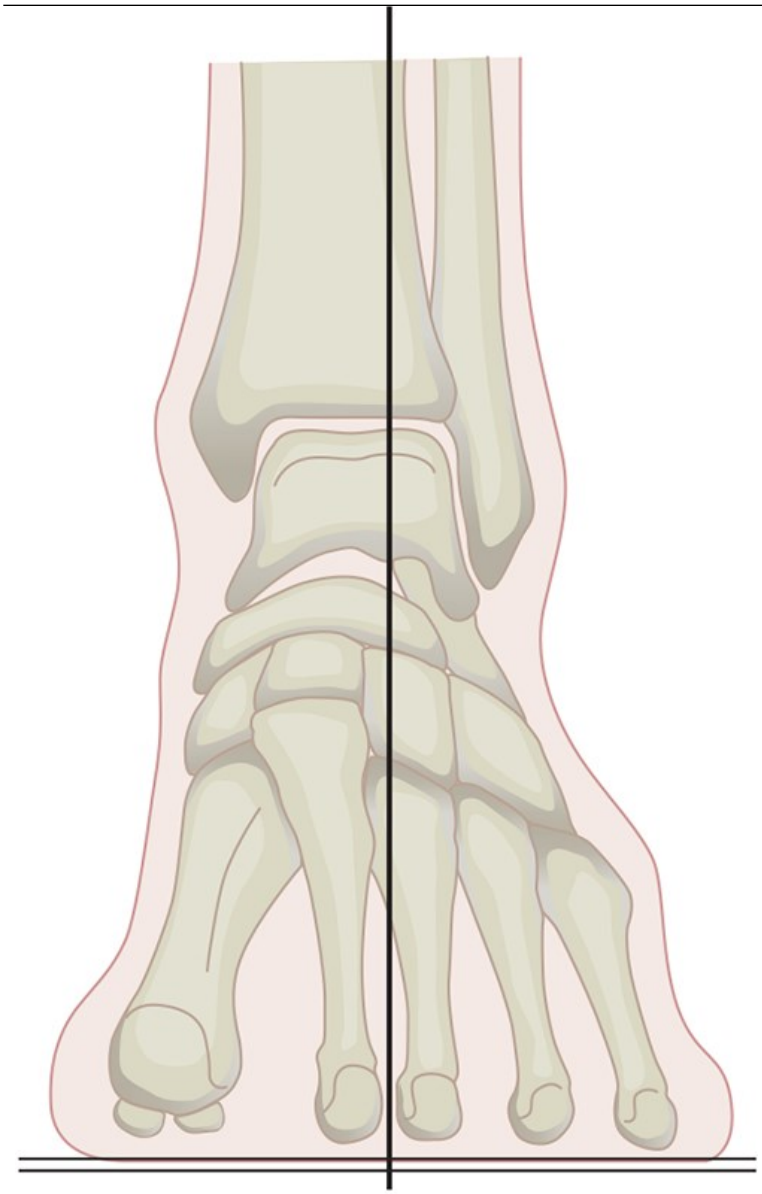

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Various anatomical abnormalities or pathologies in the neuromuscular system can create imbalances in the musculoskeletal system. These imbalances necessitate an adjustment or correction in position by either reflexive or volitional muscle action in order to realign the skeletal elements and restore balance to the system. When skeletal malalignments or restrictions cannot be corrected by muscle action, the foot must accommodate to the malposition, and the body must adapt its balance mechanisms accordingly.

The individual foot adjusts position by complex muscle and postural reactions in an attempt to maintain balance around a COG or force that is located just lateral to the center of the second cuneiform. When functioning together, the two feet and upper body postural reactions work to maintain the COG over a point located directly between the feet. During single limb support, changes in terrain or perturbations to the body cause a shift or unbalance to this center of force in one of eight directions or vectors of imbalance (**FIGURE 7-32**).[29]

FIGURE 7-32

The eight vectors of foot imbalance Four of these directions consist of the cardinal vectors of anterior, posterior, medial, or lateral. The remaining four vectors consist of anteromedial, anterolateral, posteromedial, and posterolateral. (Used with permission from Dr. Alan Whitney.)

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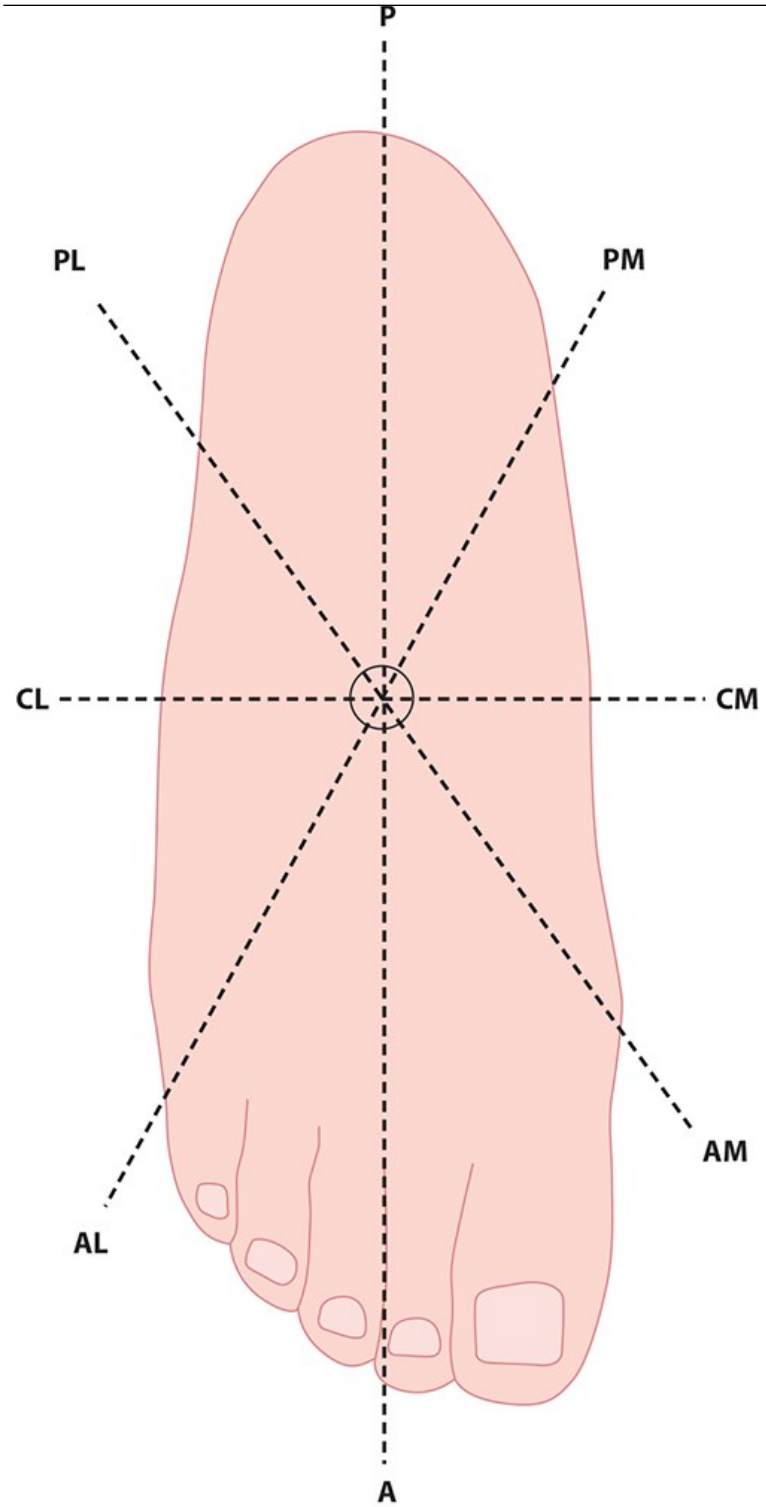

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Postural challenges caused by terrain, activity, or skeletal malalignments cause the center of force to shift in one of these eight directions; the body reacts to restore balance to the system. For many years the medical community looked at the foot using a model proposed by Merton Root.[30] In this model, the foot functioned primarily in response to various frontal plane morphologies (such as forefoot or rearfoot varus/valgus deformities or variations from an ideal normal). The subtalar joint that has a triplanar, pronatory/supinatory axis was the primary adapting articulation, and the compensations noted in each of the planes were the direct result of primary malalignments in the frontal plane. This was a useful clinical model for developing frontal plane posted foot orthoses but did not account for the planal influence of malalignments in the sagittal plane or the deforming

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forces that could be generated by rotational deformities in the transverse plane. Using a Rootarian approach, single or multiple ray deformities in the sagittal plane would be interpreted as frontal plane abnormalities, that is, a plantarflexed first metatarsal would be interpreted as forefoot valgus. This would lead the clinician to treat the patient with a forefoot valgus post; however, the intervention needed for proper balance was a heel elevation and accommodation for the plantarflexed metatarsal within the orthotic shell and the forefoot extension. The weaknesses of Rootarian biomechanics led to the development of alternative foot models that attempted to account for malalignments in all three planes (triplanar). Several experts (notably those of Alan and Ken Whitney[29] and Howard Danenberg[31]) felt that the primary focus of balance lay in the sagittal plane.

The foot has a center axis of balance or "spine," which generally corresponds to the second or sometimes the second and third metatarsals with their accompanying cuneiforms. Medially, the first ray or metatarsal-cuneiform segment and laterally, the fourth and fifth rays or metatarsal-cuboid segments function as "dynamic outriggers" by virtue of their independent supinatory/pronatory axes and their strong independent muscle attachments. If the COG deviates from the center axis, the muscles of the outriggers can be activated to exert a corrective force and thereby restore central balance. Medial shifts in balance (pronation) would be resisted primarily by contractions of the anterior and posterior tibial muscles, and lateral imbalances (supination), resisted by the peroneals. Forward shifts in the COG can be corrected by contraction of ankle and digital flexors and is noted clinically as active gastrosoleus and digital flexor contractions. This action is observed as digital gripping (**FIGURE 7-33**). Posterior shifts in balance are resisted by the dorsiflexors of the ankle and toes and are noted clinically as a retraction of the digits and visible contraction of the tibialis anterior muscle (**FIGURE 7-34**).

FIGURE 7-33

Digital gripping with anterior imbalance (Used with permission from Dr. Alan Whitney.)

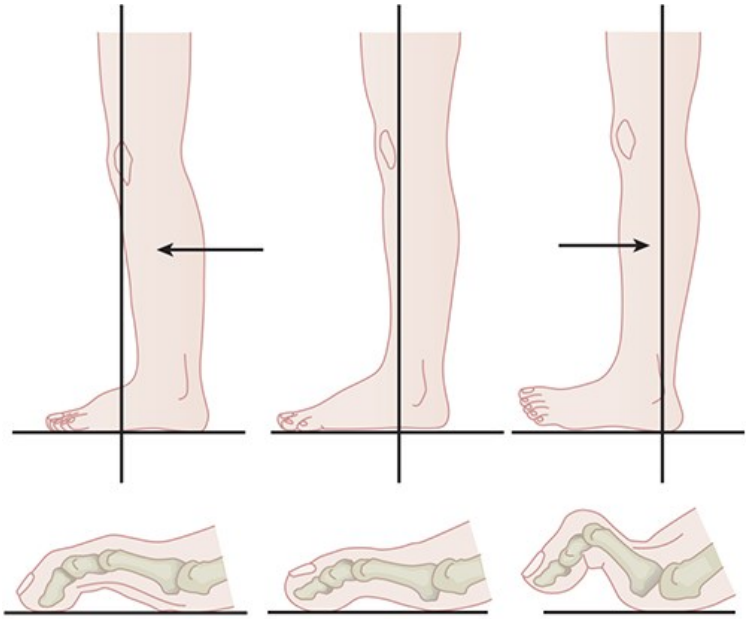

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FIGURE 7-34

Intrinsic muscle wasting with retraction of digits 2–5

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Skeletal malalignments that produce a permanent imbalance by virtue of their axis orientation and subsequent compensations are stabilized by these same muscle groups; however, they are seen as positional deformities that are initially flexible in children but progress to fixed deformities with age.

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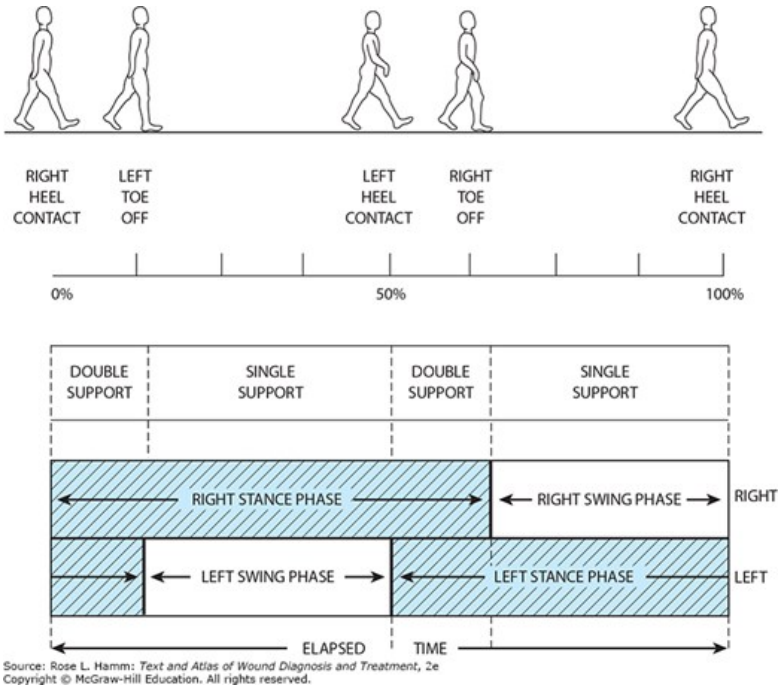
The deformities are often accompanied by degenerative arthritis due to articulation overload in one direction or another. For example, chronic anterior imbalances produced by forefoot varus or valgus or hypermobile ray segments result in relative dorsiflexion of the forefoot on the rearfoot, thereby producing a chronic digital gripping that is referred to as flexor stabilization of the toes. This alignment leads to hammertoe deformities with subsequent development of digital corns and calluses as a protective reaction to pressures caused by shoes or ground reactive forces.[32] Chronic posterior imbalances are observed with rigid plantarflexed rays and produce sagittal plane compensations, eg, retracted digits with resultant dorsal corns and plantar calluses due to loss of digital purchase and excess pressures that are created by exposed plantarflexed metatarsals. Any plantarflexion of the forefoot on the rearfoot must be compensated either by a segmental elevation of the affected ray(s) or by a dorsiflexion of the forefoot at the ankle. Any dorsiflexion of the ankle produces a tightening of the posterior muscle group or a reduction in available motion at the ankle joint, a term referred to as *pseudoequinus* by Green and Whitney.[33]

Jacquelin Perry divided the sagittal gait cycle into three rockers or pivots essential for normal gait.[34] At heel strike the calcaneus strikes the ground and the body rolls over the relatively rounded plantar calcaneal tubercles, which is the first rocker. As the foot moves into midstance and comes into full contact with the ground, sagittal forward movement is transferred to the second or ankle rocker. This pivot allows the leg to move forward over the now planted foot (**FIGURE 7-35**). The Achilles tendon then tightens and the posterior muscle group contracts in order to resist the rapid forward movement of the tibia over the ankle and thereby stabilize or "lock" the midtarsal joints. These muscles exert a plantarflexory force transferring motion to the third or forefoot rocker for push-off. As the body is propelled forward, the heel lifts from the ground and the ankle plantarflexes, causing the body to pivot over the metatarsal heads. Relatively small pronatory and supinatory drifts are noted as the foot progresses through a normal ideal gait cycle: pronation or anteromedial imbalance at heel strike, resupination or anterolateral imbalance during midstance, and then an anteromedial shift to the first ray in the propulsive phase. The dynamic outrigger activity of the muscle groups is generally eccentric to resist exaggerated movement in the direction of these imbalances and to act as a force couple around the virtual center axis of balance. This activity produces a stable foot and a smooth transfer of forces between each of the rockers, thereby leading to a graceful appearance to the gait.

FIGURE 7-35

Normal gait cycle



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The midfoot, and particularly the midtarsal joint, acts as both a flexible shock absorber at heel strike into midstance, and a rigid lever for push-off. Several mechanisms are used to accomplish this. The joint axis orientation of the subtalar, talonavicular, and calcaneocuboid joints allows for the foot to absorb pronatory forces by talar medial rotation at the subtalar joint. It also allows the forefoot to dorsiflex on the rearfoot at the midtarsal joint. Subsequently during resupination, these same joints, aided by their axis orientation, move into a position of relative stability. This positioning is also aided by derotation of the tibia as a result of forward motion of the opposite swing limb, passive tightening of the plantar fascia during heel rise

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utilizing the *windlass mechanism*,[35] and dynamic stabilization by concurrent contraction of the posterior tibial and peroneus longus muscles that effectively locks the midtarsal joint. The importance of dynamic stabilization has generally been downplayed in Rootarian biomechanics, favoring the more passive locking of the midtarsal joint caused by the crossing axes of the talonavicular and calcaneocuboid joints.[30]

Many people function in a relatively pronated position during push-off because of various forefoot and rearfoot planal malalignments related to their inherited foot types. Unless there is a neuromuscular weakness or control problem, the dynamic activity of the peroneus longus and posterior tibial muscles is adequate to stabilize the midtarsal joint in a number of pronated or supinated attitudes, and thus allow for efficient transfer of forces to the forefoot at the time of propulsion.[35] Inability to exert muscle force during propulsion results in a transfer of supportive responsibility from the muscles to the ligaments and passive support structures of the foot. If they are inadequate to resist the forces of weight plus momentum, the structures will either give way (tear) or attenuate over time, resulting in a midfoot collapse (**FIGURE 7-36**). Atrophy and loss of power occur over time as a result of diabetes, thereby resulting in loss of stability of the foot and a fixation of the deformities due to collagen cross-linking (see the section "Glycosylation").

FIGURE 7-36

Midfoot collapse

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When these imbalances cause excessive medial or lateral translations of the center of force, and the muscles designed to correct for these translations are either at a mechanical disadvantage or compromised by weakness or paralysis, marked deformities can develop. If they are allowed to persist throughout life, they become fixed deformities.

This discussion of normal foot biomechanics is important in order to understand the diabetic foot. The diabetic foot is slowly compromised by the intersection of several component causes of ulceration.[12] These include the classic triad of neuropathy, deformity, and repetitive trauma.[36] It also includes vascular disease, footwear, lifestyle, activities, and compliance or adherence issues. These can be represented graphically by intersecting spheres (**FIGURE 7-37**). As these various factors intersect in different combinations, the demands and stresses placed on the diabetic foot can exceed its capacity to adapt and ulceration or mechanical dysfunction can ensue.

FIGURE 7-37

The intersection of component causes of the diabetic foot wound

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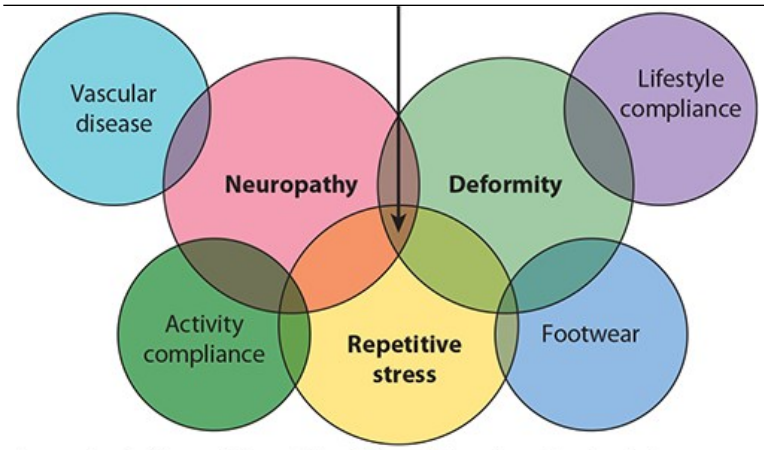

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In the classic diabetic foot model, neuropathy leads to ulceration because of the inability to feel trauma and resultant ulceration at points of excess pressure and shear. Patients with diabetes have been shown to break down in areas of both high and low pressures.[37] Simple explanations are often the best to help understand complex problems, but a simple explanation for diabetic ulceration often leads providers and payers to believe that simple solutions should suffice. In reality, a much more complex and comprehensive approach is required. It cannot be assumed that only sensory neuropathy is important with diabetic foot function. Joint position sense is just as important as skin sensation, as well as more proximal balance disturbances noted with a loss of distal proprioceptive input. When the persons with diabetes cannot feel either pain or position, they are apt to compensate by using a series of destructive behaviors. These behaviors include pathologic gait alterations *or* buying short or tight shoes so they can feel the shoes on their feet. They tend to lace their shoes too tight for the same reason. They develop a wider stance and a slower gait to compensate for balance losses related to muted proprioceptive input and delayed muscular balance activity. They also will develop a hard heel strike and excessive digital gripping in an attempt to increase their ground perception during stance and gait. This adaptation also serves as a recruitment phenomenon when midfoot dynamic stabilization (by the peroneals and posterior tibial muscles) is delayed or inadequate to provide a rigid midfoot for propulsion. This results in increased shear forces present on the plantar surface, exacerbating callus breakdown, and subsequent ulceration (**FIGURE 7-38**).

FIGURE 7-38

Dyshidrosis with pre-ulcer secondary to shear callus on the hallux IPJ

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All aspects of the sensitive balance and control mechanism discussed above are either restricted or weakened by a progressive stiffening of the connective tissues, gradual weakening of the muscles with subsequent imbalances, and a loss of input from sensory nerves in both the skin and proprioceptors of the foot and ankle. The abnormal gait finding of late mid-stance pronation of the subtalar joint has been proposed as a reason for many pathologies induced by biomechanics, including plantar fasciitis, intermetatarsal neuroma, hallux limitus, hallux valgus, sesamoiditis, and low-back pain.[38]

All of these entities produce pain and discomfort with resulting compensatory changes in foot position and gait in order to reduce or eliminate the discomfort. *With diabetic sensory and proprioceptive neuropathy, these pathological overloads do not produce pain and thus the patient does not initiate protective compensations.* As a result, midfoot breakdown, boney overload, and even Charcot arthropathy may ensue.

Charcot arthropathy is a particularly devastating complication of diabetes that results in multiple foot fractures and a complete loss of skeletal architecture when allowed to progress without treatment (**FIGURE 7-39**). There are three theories as to why pathological fractures develop in the neuropathic foot. The neurovascular, or French, theory suggests that increased blood flow from autonomic neuropathy leads to a “washing out” of the mineral supports of the osseous structures and a resulting osteopenia, and subsequent fracture.[39] The German theory by Virchow and Volkman proposed progressive microneurotrauma from a mechanism similar to that described above, which leads to osseous inflammation and progressive micro-fractures, resulting in macro-fractures and eventual loss of osseous integrity.[40] The third or combined theory recognizes the contribution of both mechanisms and concludes that they contribute equally to the formation of the disorder.[41]

FIGURE 7-39

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X-ray of severe Charcot bone destruction with multiple fractures and dislocations

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Glycosylation

Assessment for limited joint mobility should be part of any diabetic examination. Usually the hands are inspected using the Prayer Sign or the Table Top Test. The Prayer Sign is performed by placing the hands together in the prayer position and observing whether the patient can get the fingers and palms together. An inability to press the palms and fingers together indicates a flexion deformity of the interphalangeal and metacarpal phalangeal joints due to limited joint mobility.[42] The Table Top Test is performed by asking the patient to place the hands flat on the table top and noting the ability of the hand to lie flat on the surface.[43] Although there is no similar test for the foot, the presence of equinus as measured by the Silfverskiold test and observation of the digital position in stance can be a good substitute. According to *Wheeless' Textbook of Orthopedics*, the Silfverskiold test is performed using a goniometer to measure the degree of ankle dorsiflexion available in both the knee straight and knee bent positions (**FIGURE 7-40**).[44] With the knee straight the measurement is primarily looking at the gastrocnemius muscle, and should be 10° or more in the normal condition.

CLINICAL CONSIDERATION

Clinical screening for diabetic muscle wasting can be performed by observing the thenar eminence. Atrophy of the intrinsic muscles that form the thenar eminence may be a sign of diabetic motor neuropathy caused by median nerve compression.

FIGURE 7-40

Measurement of ankle dorsiflexion knee bent—Silfverskiold test With the knee straight the measurement is primarily looking at the gastrocnemius muscle, and should be 10° or more in the normal condition. With the knee bent the gastrocnemius muscle, which originates above the knee joint, is placed on slack and the measurement is an approximation of the tension in the soleus muscle itself. Thus with the knee bent, there should be an additional 10° of dorsiflexion or 20° total motion. If there is a restriction in dorsiflexion in both positions, then both components of the gastrosoleus complex can be assumed to be affected. (Used with permission from Dr. Alan Whitney.)

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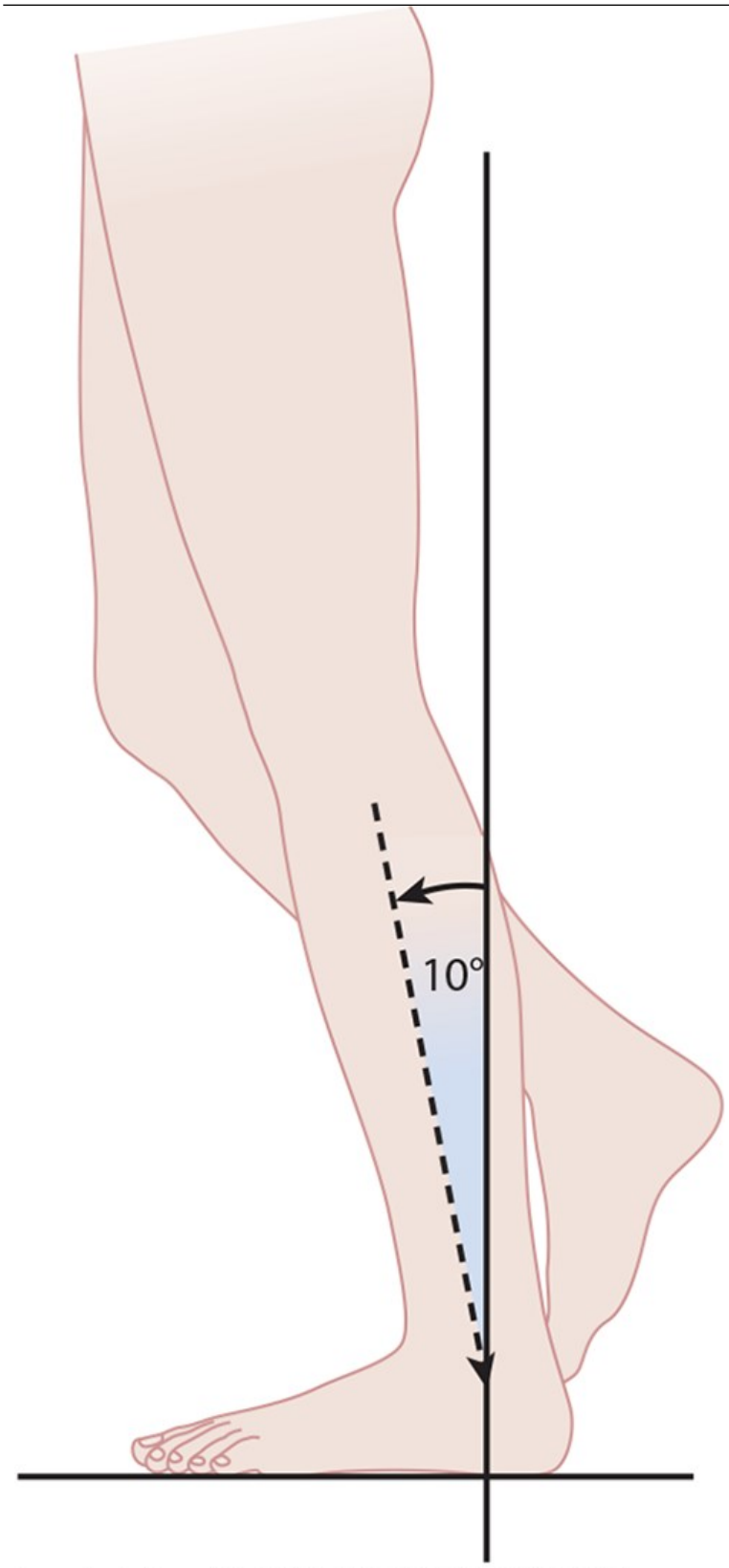

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With the knee bent the gastrocnemius muscle, which originates above the knee joint, is placed on slack and the measurement is an approximation of the tension in the soleus muscle itself. Thus with the knee bent, there should be an additional 10° of dorsiflexion or 20° total motion. If there is a

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restriction in dorsiflexion in both positions, then both components of the gastrosoleus complex can be assumed to be affected. Because of intrinsic muscle wasting associated with diabetic neuropathy, the toes are usually in a flexed or hammered position. When this condition exists for several years, the digital positions can become fixed or contracted, thereby making the assessment of a loss of flexibility due purely to connective tissue cross-linking difficult.

Long-standing hyperglycemia leads to a state where glucose reacts with proteins to form *advanced glycosylation end products* or AGEs. These products facilitate the formation of irreversible cross-links with collagen.[45] Along with the changes from AGEs, connective tissue changes associated with diabetic nephropathy, serum lipid peroxide, and dyslipidemia lead to a general stiffening of connective tissues in the body.[46] The result is a loss of flexibility in the foot and ankle complex with increased pressures generated across the forefoot, and gait-related changes that increase the risk for ulcer development. This risk increases the longer the person has diabetes regardless of the blood glucose levels.[47]

Diabetic neuropathy not only affects the sensory nerves of the patient but the entire neuromuscular system. The annual decline in ankle strength associated with diabetic neuropathy was shown to be 3% ± 2.5%.[48] This is a progressive weakness affecting both the intrinsic and extrinsic muscles of the foot. Unlike Charcot-Marie-Tooth disease, diabetic neuropathy has a propensity for the tibial nerve over the peroneals.[49] Distal intrinsic muscles are affected first with production of the classic “intrinsic minus foot.” This deformity is seen when the intrinsic stabilizers of the metatarsophalangeal joints are weakened and the long flexors and extensors of the toes unopposed.[50] Loss of the function of the lumbricales and interossei of the foot leads to hammering deformity of the digits, a loss of digital stability in all planes, a loss of distal digital purchase, anterior migration of the plantar fat pad with the retracting toes, and exposure of the metatarsal heads to excessive plantar pressures.[51]

The subsequent loss of flexibility and muscle weakness due to glycosylation and diabetic myopathy produce a foot that cannot protect itself by compensatory motions and positions, skin that ulcerates because it cannot withstand the shear forces produced by the aberrant biomechanics, and ulcerations that cannot heal due to the muting of the healing cascade produced by glycosylation of the cells necessary for a robust healing response. The ability to heal is also frequently compromised by some degree of peripheral arterial disease.

The Neuropathic Foot Wound

The process of developing a diabetic wound is a complicated one and is not the result of any one contributing factor. Tissues are initially compromised by the presence of neurovascular dysfunction and glycosylation of the connective tissues. The combination of lack of sensation, biomechanical imbalances, and unrelieved repetitive stress leads to soft tissue compromise, particularly in areas of high shear and pressure (**FIGURE 7-41**). Unrelieved pressure or infrequently relieved pressure produces a localized occlusion reperfusion tissue injury, which leads to cell damage and eventual cell death. Small areas of localized necrosis connect to form larger areas of necrosis, subsequently stimulating a localized inflammatory response.

FIGURE 7-41

Neuropathic ulcer under the cuboid after partial foot amputation of the fifth ray and the neuropathic ulcer under the medial midfoot after first ray amputation Note the severe claw toes with fat pad migration and dropped metatarsal heads where increased pressure would also occur with weight-bearing, especially at the second and third ones.

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This inflammatory response is detectable by skin temperature sensors (**FIGURE 7-42**). Areas where pathological inflammation are present will have an increase in temperature of ≥4°F in the area assessed when compared to the same site on the uninvolved foot. This sign is indicative of a pre-ulcerative callus or an underlying Charcot joint. Inflammation will be accompanied by the formation of pools of transudate between layers of the epidermis or between the epidermis and dermis which will be evident with callus debridement as maceration between tissues layers. These weakened tissues are unable to resist the normal shear forces produced during ambulation, thus leading to further tissue breakdown and fluid accumulation similar to that noted with a blister. Deeper tissue damage is accompanied by capillary rupture and blood in the tissues. This subcutaneous hemorrhage is evident as a dark staining of the tissues beneath the surface or frank blood under the callus. If the patient continues to walk on the fluid pocket, the fluid will be pushed from side to side by shear forces and dissect into neighboring areas where there is less resistance to fluid flow (**FIGURE 7-43**). When these tissues rupture through the epidermis, the protein rich transudate and blood are exposed to surface bacteria and easily become infected. The depth of tissue damage, the degree to which diabetes has compromised the patient, and the aggression of the microorganism determine the depth of ulceration. Continued trauma will only deepen the ulceration. Once an ulcer has formed, the patient with diabetes is at a distinct disadvantage when trying to heal the wound. The mechanisms by which the normal cellular response to wound healing is compromised are listed in **TABLE 7-12**.[52]

FIGURE 7-42

TempTouch temperature sensors used to detect areas of inflammation and risk for ulceration

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FIGURE 7-43

Dissecting sublesional hematoma

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TABLE 7-12

Impaired Cellular Function in Diabetes

- Impaired neutrophil and macrophage function
- Excessive deposition of matrix proteins (collagen and fibronectin)
- Fibroblastic growth factor receptors decreased
- Decreased endothelial cell response to angiogenic stimuli
- Interference with cell communication and need for keratinocyte migration
- Decreased keratinocyte migration
- Failure of timely and rapid wound contraction
- Impaired endothelial function (nitric oxide)

Off-loading the Diabetic Foot Wound

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Transitional Off-loading

The key to healing the *uninfected* diabetic foot wound is off-loading. Lack of sensation will tempt the neuropathic patient to continue to weight-bear on the wound, thus causing repetitive trauma to the delicate tissues and preventing the wound from healing. Off-loading devices shift pressure from areas of high pressure to areas of low pressure. Using a number of different mechanisms, these devices redistribute plantar pressures and reduce shock and shear forces that lead to tissue breakdown and delay the healing process.[53,54]

No single off-loading device is capable of reducing pressure on pedal wounds for the entire healing period. Dr. James McGuire introduced the concept of *transitional off-loading*[55] to describe the process of using different off-loading devices at different times during the phases of wound healing. In order to assess the mechanical risk for ulceration and develop a plan to treat an open ulcer, McGuire proposed a 6W assessment approach. The six Ws are based on the "who-what-when-where-why and how," or in this case, "way," of inquiry (see **TABLE 7-13**).

TABLE 7-13

Six-W Biomechanical Risk Assessment

Six-W Biomechanical Risk Assessment	0	1	2	Total
Who	No neuropathy or deformity (0)	Neuropathy or deformity (4)	Both (7)	
What	Properly offloaded	Adequate offloading but not ideal	Inappropriate footwear or behavior (barefoot)	
When	Limited or no ambulation	Moderate or normal daily activity	Highly active	
Where	Indoor Limited walking on uneven surfaces	Moderate outdoor walking on some uneven surfaces	Frequent outdoor walking on multiple uneven surfaces	
Why	Compliant Highly motivated	Mostly compliant Average motivation	Noncompliant unmotivated	
Way	Short stride Slow, shuffling gait	Normal stride cadence and step length	Long stride Long step length fast, hard walker	

Low Risk: 0–3, Moderate Risk: 4–6, High Risk: 7–12.

Who the patient is includes the intrinsic physical characteristics such as foot type, presence of deformity, severity of the diabetes, or the presence of complications such as renal disease. **What** the patient wears describes the choice of footwear and/or orthotic history. **When** the patient walks is an assessment of the amount of walking a patient does during a typical day. **Where** the patient walks describes the activities the patient engages in and the type of surfaces involved in these activities. **Why** the patient walks involves the motivation to adhere to the recommended amount of ambulation. How or the **way** the patient walks is described by the step and stride length and the aggression of ground strike. A relative risk scale was developed with each of the above variables placed on a grid and given a relative numerical weight to determine the *six-W biomechanical risk assessment* score for that patient. The higher the relative score, the greater the risk of tissue damage and the more aggressive the approach to off-loading must be.

Used with permission from McGuire J. Transitioning from open wound to final footwear: off-loading the diabetic foot. *Podiatry Today*. 25(9), September 2012.

Normal wound healing progresses with an orderly transition from one phase to the next. The off-loading needs are different for each stage of healing.

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Most wound care practitioners tend to use one off-loading device throughout the healing process and then have the patient return to the pre-wound footwear, which explains the high rate of recurrence after wound healing. The component causes of ulceration unique to each patient determine the various therapies that will be utilized to manage the diabetic wound.[12] Neuropathy is the most determinant factor in wound development.[56] The most common pathway to the development of a DFU is a combination of neuropathy, deformity, secondary callus formation, and elevated peak pressure.[57] Elevated foot pressures contribute to the development of an ulceration[58]; however, it has been difficult to determine if there is a specific threshold pressure that can predict risk of ulceration.[59] Frequently, the foot type and resultant compensatory changes are secondary to neuropathy as the primary reason for the development of ulcers in diabetic patients.[60] Biomechanical compensations such as equinus, hammer toes, and hyperpronation become fixed with prolonged glycosylation,[61] thus increasing the incidence and degree of skin irritation that results from shoe and plantar pressures and shear damage. Loss of digital purchase during ambulation leads to increased forefoot plantar pressures on the metatarsal heads compounded by the almost universal presence of equinus in the diabetic foot.[62,63]

Autonomic neuropathy produces dry skin that cracks easily and is more vulnerable to bacterial and fungal invasion.[64] Calluses form quickly, and without early detection can become limb-threatening problems. Off-loading should be designed to protect the foot from developing calluses and their subsequent ulcerations or at least slow their formation so they can be controlled by regular podiatric foot care.

As much as the clinician would like to blame the patient's choice of footwear for the problems presented by the patient with diabetes,[65] shoes are a poor predictor of wounding without an accompanying foot deformity. Therapeutic footwear, however, has been shown to reduce the incidence of foot ulceration.[66] Simply writing an off-loading prescription is not enough and the practitioner must counsel the patient regarding the amount of time they spend on the feet each day. Alterations of patient routine can have a significant effect on reducing the accumulation of pressure and shear due to unorganized activity. Walking on rough or uneven surfaces greatly increases shear forces and the risk of ulceration.

Repetitive activities such as treadmill exercise or daily walks for aerobic health need to be addressed when attempting to alter a patient's biomechanical environment. The patient's decision to cooperate with the treating clinician is the single most important factor in determining the success of wound healing (**FIGURE 7-44**). Without that, all efforts will ultimately fail no matter how expertly they are presented. To be successful, patients must understand *why* their clinician wants them to follow a certain treatment plan and *what* the consequences will be if they choose to deviate from that plan.[67] As clinicians, we have the tendency to expect patients to respond positively to our fact-filled presentations. This is a *provider-centered* and not a *patient-centered* approach to care.[68] It is important to treat the whole patient and not just the hole in the patient.[69]

FIGURE 7-44

Adherence This patient assured us she had remained non-weight-bearing the previous week.

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Off-loading Devices

Off-loading the diabetic foot requires a transitional approach to the many devices available to the clinician. The total contact cast (TCC) is considered the gold standard for off-loading a diabetic foot wound, by virtue of its ability to produce healing rates as high as 90% (see **FIGURE 7-16**).[70,71] The International Working Group on the Diabetic Foot, however, has concluded that relatively few practitioners use this modality on a routine basis despite the overwhelming research data to the contrary.[72] Reasons for this include established practice habits, fears of injury from the casts themselves, lack of training in its application, previous negative experiences with the device (usually by inexperienced practitioners), and reimbursement issues.[73,74] There are some instances where a patient should not be treated with a TCC. These include patients with documented PAD or an ABI of less than 0.7, fluctuating limb edema, or an active infection of the skin or the wound.[75] Other contraindications include cast claustrophobia, documented nonadherence, a sinus tract with deep extension into the foot, or inadequate training of the clinical staff in the use of the technique.

A number of alternative devices are available to off-load the DFU.[53] These include the removable cast walker (RCW) (**FIGURE 7-18**), nonremovable cast walker or instant TCC (iTCC) (**FIGURE 7-17**), D'Arco wound healing shoe (**FIGURE 7-21**) molded or double upright ankle foot orthosis (AFO) with or without a patellar tendon-bearing addition (**FIGURE 7-58**), Charcot restraint orthopedic walkers (CROWs) (see **FIGURE 7-24**), modified Carville healing sandal or shoe (see **FIGURE 7-25**), adhesive felted foam to off-load the wound in various off-loading devices (see **FIGURE 7-20**), the Rader football dressing (see **FIGURE 7-19**), and commercial off-loading shoes, such as wedge shoes (**FIGURE 7-45**), a commercial off-loading shoe with pixelated or segmented innersoles (**FIGURES 7-46** to **7-48**), or even depth or custom-molded footwear.

FIGURE 7-45

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Orthowedge shoe

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FIGURE 7-46

Diabetic healing shoe with pixelated insole

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FIGURE 7-47

Hexagonal pixelated insole

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FIGURE 7-48

Diamond-shaped pixelated insole

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Devices are selected based on clinician experience, device availability, patient pressure or preference, or even insurance reimbursement with the most commonly employed device being the surgical shoe with or without internal shoe modifications.[54,76] Shoe-based devices have relatively poor evidence for healing when compared to the TCC; therefore, they are recommended for the transition period between initial closure of the wound and the use of the patient's final footwear. Other than the TCC, the only other methods that have been shown to produce healing rates as high as 80% and thus considered a reasonable substitute are the iTCC, football dressings, and the felted foam technique.[77–79]

The key to healing with any of the off-loading devices is the clinician's ability to improve patient adherence by making it impossible to remove the device without the clinician's approval. Knowles and Boulton found that when patients were given specialized footwear free of charge, only 20% of the patients actually wore the shoes.[80] Armstrong et al. found that when given the ability to remove the devices, RCWs were worn only 28% of the time during activities of daily living.[81]

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Improving the Removable Walker

Both the felted foam and football dressings can be used with RCWs, thereby increasing the effectiveness of the devices and allowing the bulky cast to be removed at night for sleeping (**FIGURE 7-23**). Removable devices allow the clinician easy access to the wound during the healing process making it possible to apply advanced dressings to facilitate wound healing. However, any time a device can be removed by a patient there will be temptation to remove the device for comfort and thus the patient will probably walk without it.[82] A number of RCWs have been made with patellar tendon bearing (PTB) uppers or calf attachments in an effort to redistribute more weight from the foot. These devices are now available off the shelf and have only a slight edge over a standard RCW in the ability to off-load the foot. The newest type of device to be employed is the limb-load walker that uses a tight calf attachment to off-load the foot by transferring weight from the foot to the leg. These include the Torch Shoe and the Zero G brace (**FIGURE 7-49**). Once the tight upper attachment loosens because of edema reduction or weakening of the Velcro closure, the device is no better than a standard walker.

FIGURE 7-49

A. Zero G Brace with off-loading insole in place. **B.** Zero G Brace with off-loading insole removed.

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Felted Foam and the Football Dressing

Felted foam dressing (FFD) utilizes a 1/4-inch adhesive felt pad with an aperture for the wound and is applied directly to the foot to reduce pressure on the ulceration (**FIGURE 7-50**). The wound is appropriately dressed, the felt applied, and a wrap of gauze or self-adherent wrap used to anchor the felt. The foot is then placed in one of several off-loading shoes or walkers to be used during ambulation. The pads are reapplied weekly or biweekly with the selected wound dressing material until the wound is healed. Birke et al. compared FFDs with a TCC and found that 93% of the ulcers treated with the FFD were healed within 12 weeks (mean time to healing of 20.9 days) compared with 92% (31.7 days) in those treated with the TCC.[79]

FIGURE 7-50

Felted foam dressing over a cuboid ulcer

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The football dressing can be used alone or in conjunction with the felted-foam technique. It is an excellent choice for cases where a TCC is contraindicated or a cast walker cannot be obtained because of insurance limitations or other circumstances. The football dressing uses several layers of cast padding, secured with woven gauze roll bandage, overlaid by more padding, additional gauze, and finally a layer of self-adherent wrap to keep it in place (**FIGURE 7-19**). These dressings, although they are non-removable, alleviate the feeling of claustrophobia some patients experience when they are restrained in a non-removable device such as the TCC.

Transitional Devices

Recently epithelialized scar tissue cannot withstand the stresses of normal activity or the loading problems produced by the patient's original footwear. Steed et al. found that 69% of patients broke down within 30 months after closure with a growth factor.[83,84] Pixelated innersoles or custom-made total-contact molded innersoles in footwear such as a surgical shoe with a rocker sole (**FIGURE 7-51**) or a prefabricated healing shoe (**FIGURE 7-21**) are excellent transitional devices to prepare the patient for ambulation in the final footwear.

FIGURE 7-51

Surgical shoe with total contact molded insole

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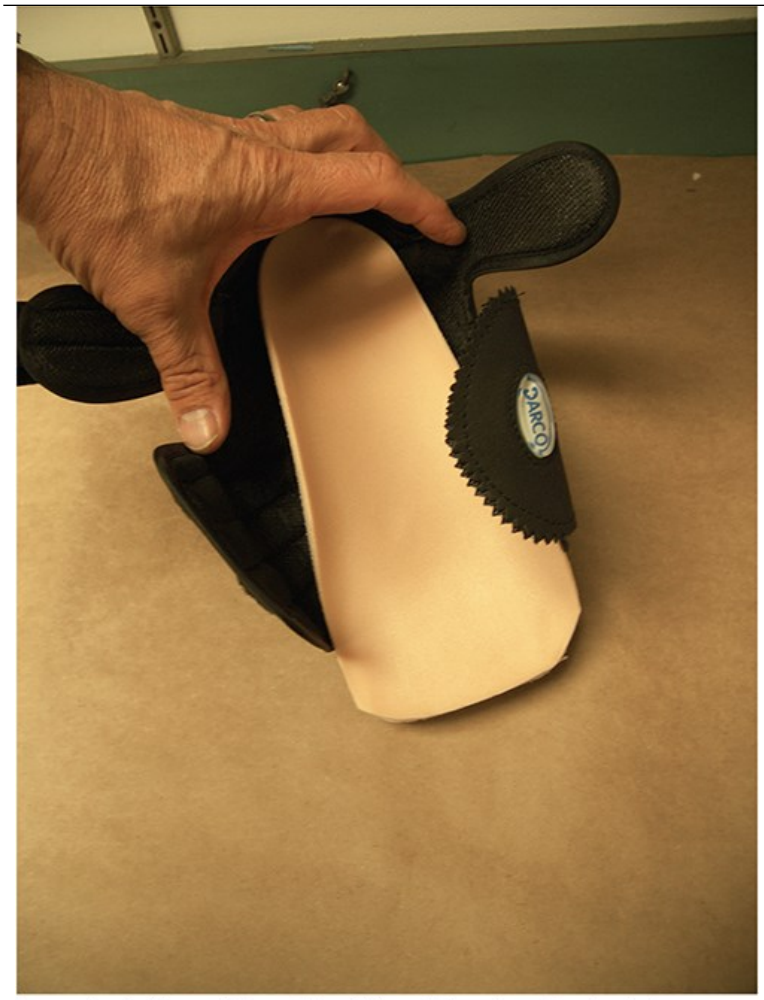

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Wound Care

Wound care for the DFU follows the TIME paradigm presented in Chapter 13 and consists of debridement of necrotic tissue, including the periwound callus that consists of senescent cells that will inhibit wound closure; treatment of infection with topical antimicrobial dressings and systemic antibiotics when indicated; moist wound dressings, beginning with absorbent dressings sufficient to manage the drainage and protect the off-loading device from moisture that will facilitate more bacterial growth; and close attention to the edges to facilitate re-epithelialization as the wound granulates. Numerous references are available to support the use of cellular and tissue products discussed in Chapter 13, Wound Dressings.[85–89]

A final note for wound care clinicians caring for patients with a history of recurrent DFUs—corrective surgery by a podiatrist or ankle/foot orthopedist experienced in caring for these patients can be beneficial in correcting bony abnormalities that underlie the soft tissue that is in a breaking down/scarring cycle. Surgeries such as metatarsal head resection, Achilles tendon lengthening, tibialis anterior lengthening, and flexor digitorum longus tenotomy are procedures that can alter foot structure and thereby influence gait mechanics in such a way that shear and pressure are relieved, sometimes more than can be done with diabetic shoes and inserts.[90]

Footwear for People with Diabetes

The choice of footwear must be a collaborative effort between the patient, the physician or physical therapist, and the pedorthist. All podiatric, and many allopathic and osteopathic physicians, receive specialized training in the skills necessary to diagnose diabetic foot disorders and prescribe interventions and footwear. Matricali et al. found that 67% of the patients developed recurrent ulcers 22 months after treatment with TCC treatments.[84]

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The in-depth shoe is the basis for most footwear prescriptions. It is generally an Oxford-type or athletic shoe with an additional 1/4–1/2 inch of depth throughout the shoe, allowing extra volume to accommodate any needed inserts or orthoses, as well as deformities commonly associated with a diabetic foot. In-depth shoes also tend to be light in weight, have shock-absorbing soles, and come in a wide range of shapes and sizes to accommodate virtually any foot. They also now come in a variety of styles to appeal to those conscious of footwear appearance.

According to the Medicare Therapeutic Shoe Bill, the prescribing physician is responsible for ensuring the shoes and insoles or orthoses prescribed are appropriate in both fit and function to address the needs of the patient.[91] The Medicare Shoe Bill is designed to provide diabetic footwear, total contact molded inserts, and modifications for those patients or beneficiaries who meet the criteria listed in **TABLE 7-14**.

TABLE 7-14

Therapeutic Shoe Bill for Patients with Diabetes

This bill covers patients with diabetes and covers one pair of depth shoes and three pairs of inserts or shoe modifications per calendar year.

The following criteria must be met:

The "certifying physician" **must be** an MD or DO actively managing the patient's diabetes.

The certifying physician **must document** the patient has one or more of the following conditions:*

- Peripheral neuropathy with evidence of callus formation
- Foot deformity (any type)
- A current or previous pre-ulcerative callus (intralesional hemorrhage, or maceration)
- A current or previous foot ulceration
- Amputation of all or any part of the foot
- Poor circulation
- *This document **cannot be provided by** a Podiatrist (DPM), PHYSICIAN Assistant (PA), Nurse Practitioner (NP), or Clinical Nurse Specialist (CNS) but they can sign the order for the shoes and inserts themselves

The physician must provide (**annually**) to the supplier:

- A detailed written order
- A copy of an office visit medical record indicating you are managing the patient's diabetes
- A copy of your medical office note describing one of the qualifying conditions, or an office visit note from a DPM, PA, NP, or CNS that describes one of the qualifying conditions
- The note must describe the **specific** foot deformity (bunion, hammertoe, etc.), the exact location of the foot ulcer or callus, type of amputation, and/or signs and symptoms supporting the diagnosis of peripheral neuropathy or peripheral vascular disease (venous or arterial)[92]

Data from http://www.medicarenhic.com.

It is best to refer the responsibility for the specifics of the actual shoe prescription to a licensed podiatrist because of the specialized training in biomechanics and diabetic foot care. Diabetic footwear today is available in many styles, shapes, and sizes; the pedorthist has been trained to fill shoe prescriptions for diabetic patients and has the knowledge of the market to guide patients to make appropriate shoe choices to prevent further foot injury. The patient must be educated to make wise choices with regard to footwear and lifestyle. In order to live a long and full life, the patient must embrace the basics such as eating a proper diet, monitoring blood sugar, and purchasing and wearing protective footwear, and performing daily foot checks.

There are many shoes that on the surface appear to be an adequate substitute for the real thing, but in reality do not meet the criterion for a true extra-depth shoe as defined by the diabetic shoe bill. Regardless of education to the contrary, the majority of patients will return to their pre-ulcer shoe size and style. Patients have a strong desire to return to "normal" and convince themselves that they are different from other diabetics. Even if they have developed an ulcer, they will convince themselves that this was a fluke and that they will be able to return to their former way of life without having to make major considerations. Nothing could be further from the truth and reinforcement of the need for appropriate shoes is required from all the disciplines working with the patient.

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Health care providers must educate the patient with diabetes and the caregiver that the time right after the first wound develops is the most critical time. For the open wound, the first 4 weeks of healing are the “golden hour” for avoiding the complications of osteomyelitis and amputation. In the same manner, the first several months after the first wound are a “momentum veritatis” or moment of truth with regard to the patient's future health. Patients with diabetes need to understand the gravity of an open wound. When a wound is open for more than 4 weeks, its chance of healing reduces dramatically and the risk of infection increases five times. When a wound becomes infected, the patient is 155 times at greater risk for amputation secondary to overwhelming infection or osteomyelitis.[15] After a patient develops a new diabetic wound, the 5-year survival decreases drastically with mortality rates reported between 43% and 55%.[93] After an amputation, 61% of those patients die within 5 years.[94] If patients with diabetes think they can return to previous lifestyles, they are “dead wrong”! Health care providers cannot believe or trust their assurances and soft pedal their need to control the diabetes and get into the right shoe and insert combination. The very least the provider should do is provide the proper prescription or referral for shoes as well as a reputable list of pedorthotists. In addition, the patients should receive regular podiatric foot care and periodic visits with the physician who is managing the diabetes in order to continually reinforce proper maintenance of blood sugar levels, diet, and exercise routines.

Because of the digital deformities associated with the neuropathic foot, a standard shoe is *absolutely not* an option for patients. Anatomically shaped custom molded shoes are not necessary for all patients with diabetes, and there are many styles available that provide the protection that the diabetic foot needs.

Prior to the development of an ulcer, most patients with no or low risk factors can be managed in standard footwear. After an ulcer has developed and healed, even patients with relatively minor foot deformities and low levels of risk need to be protected from future ulceration with a total contact molded innersole and extra-depth shoes with rocker soles.

Flexible foot imbalances that can be corrected by realignment are best treated with a corrective functional device to reduce the digital and forefoot compensations caused by the biomechanical imbalances. Rigid fixed deformities are best treated with an accommodative device with which areas of high pressure and shear specifically are addressed with relief areas in the innersole or soft inserts at the site of ulceration. Rocker soles are added to the shoes when there is a real risk of repeat ulcers in the forefoot and when the patient has enough balance to master ambulation with them. Rockers can be toe only for distal digital ulcers or metatarsal rockers to address metatarsal head lesions (**FIGURE 7-52**). Heel rollers or a cushion insert known as a SACH-style heel can be added to smooth out heel strike and prevent foot slap at heel contact, thus making it easier for the patient to adjust to the rocker platform (**FIGURES 7-53** and **7-54**). A double rocker sole can be used to provide additional pressure relief for patients who present with a rocker bottom or Charcot foot (**FIGURE 7-55**).

FIGURE 7-52

Commercial depth shoe with heel roller toe rocker

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FIGURE 7-53

Customized depth shoe with metatarsal rocker and a SACH

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FIGURE 7-54

SACH heel insert

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FIGURE 7-55

AFO with double rocker sole

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The concept of total contact innersoles was first introduced by Dr Paul Brand in 1983.[95] Simple flat insoles, no matter how much cushion they provide, are not adequate to off-load the diabetic foot. Insoles that have to mold to the diabetic foot during normal ambulation, a process known as dynamic molding, are also inappropriate for off-loading a foot with demonstrated risk factors for ulceration. A contoured total-contact molded innersole is essential to distribute weight-bearing forces across the entire plantar surface and reduce anterior-posterior and medial-lateral shear forces in the shoe. Providers should not allow patients to receive unmolded innersoles to save time or money. The use of a heat gun directed into the shoe for a few seconds to warm the innersoles that come with the shoes does not provide the pressure redistribution of total contact molding. Patients cannot use their at-risk feet to act as molding agents for insoles that are inadequate, and simple flat innersoles are inadequate to properly off-load a neuropathic foot. Molded insoles are thick enough to fill the midfoot arches for proper pressure redistribution and to accommodate all plantar deformities. They should not, however, in any way crowd or tighten the shoe, particularly in the area of the toes (**FIGURE 7-51**). Newer orthotics and innersoles have been designed to aggressively off-load the foot in areas where ulcers are prone to form.

Penn State University has developed an innovative method for the production of a uniquely shaped therapeutic insole to alter the plantar pressure distribution of the foot. The TrueContour diabetic insoles are presently under evaluation in an active clinical trial to determine their effectiveness in the prevention of recurrence of diabetic ulceration (**FIGURE 7-56**).[96] Rocker soles were originally proposed by Dr Paul Brand for the prevention of ulcer recurrence after total contact casting.[95] The original rocker design used a 22° metatarsal rocker with a very thick sole necessitating the use of a similar thickness insole on the opposite foot (**FIGURE 7-27**). This was very effective at off-loading the forefoot but created problems with gait and increased the fall risk for older patients. Most rocker soles today are between 12° and 18° and come in a number of designs based on the position and number of rockers on the sole (**FIGURE 7-55**).

FIGURE 7-56

Diapedia insole

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Custom molded shoes are designed to use with severe deformities that absolutely cannot be accommodated with available commercial last shoes (**FIGURES 7-28** and **7-57**). A version of a custom shoe with limb loading capacity commonly used to address an unstable Charcot foot is a *Charcot restraint orthopedic walker* or *CROW* (**FIGURE 7-24**). This rigid clamshell device is custom made to house a collapsed or collapsing foot, which needs PTB loading to reduce pressure on a delicate at-risk foot. When a patient is very heavy, additional support may be needed using a reinforced double upright AFO attached to a custom molded shoe with a PTB upper attachment (**FIGURE 7-58**). Each of these orthoses is very expensive and should only be used when standard footwear choices have been eliminated.

FIGURE 7-57

Custom molded shoe with Charcot foot

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FIGURE 7-58

Reinforced double upright AFO with patellar tendon bearing brace

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SUMMARY

The person with diabetes is a challenging, often frustrating patient to treat. Each clinician must exhibit patience, persistence, and a commitment to "do the right thing."[73] It is human to fail to comply or cooperate and it is equally human to fatigue and give up in the face of difficulty. Health care providers cannot give up or let the fatigue of a prolonged healing course prevent them from focusing on the primary goal of a healed wound and the secondary goal of prevention of recurrence. The patient's limb and life depend on it.

CASE STUDY

CONCLUSION

The vascular assessment includes trophic changes of the skin and nails, an absent pulse, and hair loss with a slightly delayed capillary filling time; these would necessitate a referral for non-invasive vascular testing that would consist of ankle brachial indices, pulse volume recordings, and toe-brachial indices. This information would indicate whether the patient would be able to heal his wounds. An ABI of 0.7 would be required for healing in a reasonable period of time (12–16 weeks). Any limitation of blood flow to the limb would necessitate a referral to a vascular specialist to determine if revascularization surgery could improve his circulation and increase his chances of healing.

The evidence of fungal infection is typical of diabetic patients but apart from oral medications that have a slight risk of liver toxicity, there are no good topical medications available and care usually consists of regular podiatric visits for nail thinning and debridement.

The neurologic assessment indicates the presence of diabetic peripheral neuropathy, which significantly increases his risk for ulceration and

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complicates the healing process. Neuropathic patients are more likely to have problems with off-loading adherence because they cannot feel pain from their wound, thereby making it less likely that they will take the wound seriously.

The biomechanical assessment is the most useful part of the examination for prescribing offloading devices, suggesting activity restrictions, and for predicting how the patient will respond to treatment. Based on our non-weight- and weight-bearing assessment, this patient ambulates on a significantly pronated foot with digital gripping making him prone to develop forefoot and digital ulcers. Equinus is a major contributing factor for elevated forefoot pressures, increased shear forces, and a transfer of forces to the midfoot, which if flexible will collapse with loading. This patient exhibits mid-tarsal joint hypermobility making him prone to this. Pronation also produces a shift of forces to the medial forefoot due to the abduction of the forefoot on the rearfoot and the anterior medial imbalance produced by the equinus and the inward collapse of the ankle during heel strike to midstance. Medial loading of the forefoot in this case leads to the development of a wound under the first metatarsal. This patient also has limited range of motion of first metatarsophalangeal joint (MPJ) known as hallux limitus. As the forefoot loads and progresses from midstance to propulsive phase, a restriction at the first MPJ would lead to a transfer of force to the hallux interphalangeal joint or IPJ. If the force transferred is limited, a pinch callus will develop at the medial aspect of the IPJ. If those forces are excessive then an ulcer may develop at the IPJ, the most common site for a forefoot wound. This medial force transfer with pronation also leads to the development of hallux abductovalgus with a medial bunion in this patient. Let us assume this patient is active and has a job that requires a lot of walking. This makes him much less likely to accept a restriction on activity from a wound he cannot even feel.

An open wound in this case necessitates the use of a non-removable off-loading device, either a total contact cast (TCC) or an instant total contact cast or iTCC. An ambulatory aid of crutches or a walker so he could remain non-weight-bearing would be best; however, at least a cane should be prescribed to slow his gait and reduce forces on the foot. If at all possible, the patient should be counseled to remain out of work for 4–6 weeks until he has demonstrated a desire to adhere to the recommendations *and* the wound has begun to heal (closed or almost fully epithelialized) sufficiently for him to transition to a removable type device. After 3 weeks in a removable walker he could transition to a healing shoe or modified healing sandal while his final footwear is being prepared by a pedorthist. Under no circumstances should this patient return to his pre-ulcer footwear. That would be an invitation to reulcerate within a very short period of time.

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